

THE LASTING IMPACT OF EARLY LIFE INEQUALITIES

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This dissertation analyzes how early inequalities in health and education affect later educational, health, and social and emotional outcomes. I place particular emphasis on how the unique economic circumstances of the developing world interact with the dynamic and multidimensional nature of child development. In Chapter 1, I build a theory of human capital formation that links early health differences to observed schooling gaps across countries. My main finding is that early health inequalities are amplified into large schooling gaps *within* and *across* countries. In Chapter 2, I use a unique Indian data set to link private school attendance to measures of self-efficacy and self-esteem and find that private school may play at least as significant a role in early psychosocial as in cognitive development. Finally, in Chapter 3, I construct a novel measure of seasonal food scarcity and find that prenatal exposure to scarcity has a lasting impact on childhood health in Ethiopia. Overall, my findings highlight the importance of understanding the dynamic and multidimensional nature of child development for effective targeting of policy interventions in the developing world.

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INTRODUCTION

This dissertation analyzes how early inequalities in health and education affect later educational, health, and social and emotional outcomes. Moreover, it centers around the view that child development is a dynamic and multidimensional process. I apply insights from the literature on child skill formation to examine how alternate facets and stages of development interact to determine economic success individually and as an economy. The level and inequality of inputs during critical stages of development may be a particular concern in economies with limited resources for later remediation and/or with substantial wealth disparities. In light of this, I place a particular focus on the lasting impact of early life inequalities in the developing world. Isolating the critical facets and stages of child development in the context of developing countries can help policymakers target cost-effective interventions towards some of the world's most vulnerable populations.

The structure of this dissertation is divided into three chapters that cover overlapping but distinct aspects of early life inequalities in the developing world. Chapter 1 begins by developing a quantitative theory of human capital to investigate the role of early childhood health in determining schooling attainment gaps *within* and *across* countries. While individuals are assumed to differ in their learning ability and access to financial resources, countries differ in public subsidies to health and education, financial development, and relative factor and human capital investment prices. My main finding is that early health inequalities are amplified into large schooling gaps *within* and *across* countries—raising investments for all children to equalize early health within countries reduces schooling Ginis by an average of

12% in developing economies and reduces the cross-country standard deviation of average schooling attainment by over 19%. A key policy takeaway is that the gains from early health interventions tend to be amplified by later educational investments in developing economies, while those targeting school-aged children may be limited if early health conditions are ignored. These findings reinforce the notion of child health policy as an effective educational policy tool.

While Chapter 1 explores the link between early health and later schooling choices in the developing world, Chapter 2 examines the impact of early educational investments on a child's social and emotional traits. In recent years, economists have paid increasing attention to the importance of social and emotional skills on a variety economic and behavioral outcomes. At the same time, there is increasing evidence that schooling is intimately linked to the development of such noncognitive traits. In Chapter 2, I examine a previously unexplored mechanism by using a unique Indian data set to link private school attendance to early noncognitive outcomes. I find that the effects of attending private school between age five and eight on self-efficacy and self-esteem are within the range of credible estimates of effects on cognitive outcomes in India, suggesting that private school may play at least as significant a role in early psychosocial as in cognitive development. Moreover, effects differ by observable characteristics such as gender, urbanization, caste, household wealth, and maternal measures of self-efficacy and self-esteem. I also find that effects may differ between children who are similar in their observed characteristics based on unobserved differences in cognitive ability, health, and social skill. These results highlight the importance of moving beyond traditional cognitive measures and average treatment effects to examine potential variation in noncognitive gains from policy interventions in the developing world.

Finally, Chapter 3 of this dissertation shifts from educational and noncognitive outcomes to explore the impact of early inequalities on later health outcomes. Specifically, I analyze the impact of prenatal exposure to seasonal food scarcity on childhood health in Ethiopia. I construct a novel measure of seasonal exposure based on reported months of relative food

scarcity in the local community. I find that exposure has a significant negative impact on height by age five that strengthen by age eight. Effects decrease with household wealth and maternal education and are stronger during the first trimester of gestation. In contrast to height, effects on child body mass are only identified closer to birth and when exposure is concentrated in the second trimester. Overall, results highlight that in addition to the effects of severe famine conditions identified in many studies, more regular variation in prenatal food availability can have lasting impacts on health in the developing world.

1. EARLY CHILDHOOD HEALTH AND SCHOOLING ATTAINMENT GAPS WITHIN AND ACROSS COUNTRIES

1.1. INTRODUCTION

Children of poor health tend to be outperformed by their healthier counterparts on educational outcomes ranging from test scores, to absenteeism, to grade levels completed (Currie 2009, Behrman 1996). These educational gaps are particularly well documented in the developing world where early childhood malnourishment is prevalent among a much larger portion of the population. At the same time, large and persistent schooling attainment gaps also exist across countries (see Figure 1.1). This implies that if early health investments impact later educational choices, they may be an important source of cross-country variation in schooling. In light of these observations, this chapter examines to what extent early childhood health differences can explain schooling attainment gaps *within* and *across* countries.

My strategy for investigating the relationship between health and schooling is to build a quantitative theory of human capital that predicts endogenous investments in early childhood health as well as later investments in education.¹ To study the influence of early health

¹By explicitly considering child health, my work builds on and complements a growing body of empirical macro literature that more broadly examines cross-country differences in human capital accumulation (e.g.

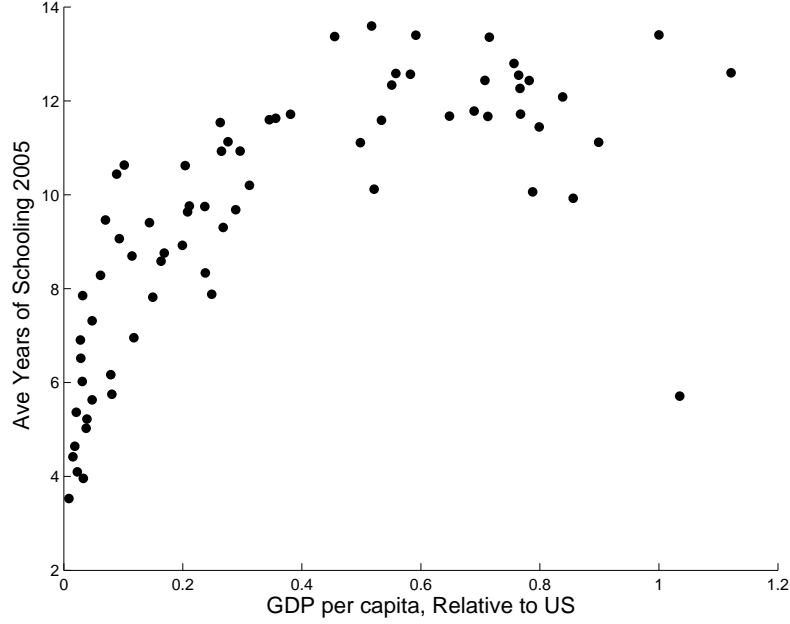


Figure 1.1: Cross-Country Dispersion of Schooling. Source: PWT 7.2 and Barro and Lee (2010) for population aged 24-29.

inequalities *within* and *across* countries, I extend heterogeneity along each dimension. Specifically, while agents within an economy differ in their available financial resources as well as learning ability, countries are assumed to differ in public subsidies to health and education, financial development, and relative factor and human capital investment prices. In congruence with the empirical evidence, I model early health and later educational investments as complements in the production of human capital. This dynamic complementarity combined with credit frictions allows for the key mechanism linking early childhood health to schooling gaps in the model. Specifically, poor financially constrained parents invest fewer resources in the health of their young children than richer parents. This, in part, results in less schooling later in childhood due to the complementarity between early health and education. In other words, early health inequalities are amplified by later educational investments.

The theory allows for quantitative analysis in a general equilibrium setting to determine the extent that early health differences ultimately impact schooling choices. Operationaliz-

Bils and Klenow 2000; Erosa et al. 2010; Manuelli and Seshadri 2014; Cordoba and Ripoll 2013; Restuccia and Vandenbroucke 2014).

ing the model relies on estimating a number of key parameters governing the production of human capital, such as the complementarity between early health and educational investments. However, because there is little micro evidence to directly pin down many of these parameters, I restrict them to match a number of cross-sectional data moments from the United States. In this manner, *within-country* heterogeneity is also helpful for empirically restricting key parameters of the model. After careful calibration, I quantitatively assess the ability of the model to account for the empirical data on schooling inequality within and across a sample of countries. Overall, the model performs quite well as the correlation between the model predictions and the data is 0.87 for country-level average schooling attainment and 0.73 for schooling Gini coefficients. The model also predicts 97% of the standard deviation of schooling attainment and 71% of the standard deviation of schooling Ginis across countries.

Quantitative analysis using the calibrated model yields two main findings. First, early health inequalities are amplified into large schooling differences *within developing countries*. Counterfactually raising investments for all children to equalize early health *within-countries* reduces schooling Ginis by 12% and increases schooling attainment by 28% on average. This occurs because the increase in early health raises the productivity of later educational investments due to complementarities, particularly for the children of poor, financially constrained parents. This amplification effect is much smaller in higher income countries where fewer parents are constrained and most children are healthy enough in the baseline to realize gains from attending school for a significant length of time. In these countries, further increases in early health levels are accompanied by smaller changes in schooling due to the decreasing returns and increasing opportunity costs of attending school.

My second main finding is that early health inequalities are an important source of schooling variation *across* countries. As a result of the differing effects across national income levels, the above experiment not only reduces schooling gaps in developing countries, it also results in more than a 19% reduction in the *cross-country* standard deviation of average

schooling attainment across the full sample. When I extend the analysis further by equating early health for all children in all countries to the *average* U.S. level, the standard deviation of average schooling attainment is 53% lower than the baseline. This occurs predominately due to a nearly 50% increase in average schooling among developing countries. In these economies, early health levels are relatively low for all children in the baseline, thus increasing to U.S. levels results in a sharp rise of the productivity of later educational investments. In higher income countries, the effect on schooling is again considerably smaller due to the already high levels of initial early health.

In addition to my main counterfactual analysis, the model is also well suited to examine the comparative effects of early health versus education subsidies on human capital investments. Specifically, I find that early health interventions in the developing world tend to have larger impacts on schooling and other outcomes than later educational interventions. In a set of policy experiments, an increase in early health subsidies increases schooling by more than double — and log-output by more than triple — a cost-equivalent increase in later subsidies to educational spending. Due to the dynamic nature of child development, increasing early health subsidies induces higher levels and less inequality in early health investments *and* later educational investments. However, while the increase in educational subsidies has a large effect on educational quality expenditures, it has much smaller effects on early health investments and schooling time. Intuitively, while poor families invest more in educational quality because it is cheaper, due to financial constraints they are unable to adjust earlier investments in health to fully take advantage of complementarities. A key policy takeaway is that gains from early health interventions tend to be amplified by later educational investments in developing economies, while those targeting school-aged children may be limited if early health conditions are ignored.

This chapter contributes to several lines of literature as it is the first to combine early health investments, dynamic complementarity, credit frictions, and heterogeneity within and across countries. A number of papers investigate the role of credit frictions when child

development is explicitly modeled as a dynamic process (e.g. Restuccia and Urrutia 2004; Caucutt and Lochner 2006; Cunha et al. 2006; Cunha and Heckman 2007; Cunha et al. 2010; Caucutt and Lochner 2012). Indeed, the human capital technology I employ is based on the theoretic work that has emerged from this research. In general, this line of literature finds evidence of strong dynamic complementarity over childhood and meaningful effects of credit constraints. However, this chapter is the first to extend the theoretic framework in a cross-country setting and to explicitly consider health investments.²

Although to my knowledge no existing models explicitly attempt to link *within-country* inequality to *cross-country* schooling differences, this chapter is related to a number of others that more generally examine cross-country differences in human capital accumulation. In the model most closely related to mine, Erosa et al. (2010) assess how human capital and productivity interact in order to explain cross-country income differences utilizing a model with heterogeneity within and across countries. However, health investments are not considered and childhood is modeled over one period—negating the effects of dynamic complementarity of investments. Although not the focus of the paper, their model does not effectively explain the distribution of average schooling attainment across countries and within-country schooling inequality is not reported outside the U.S. benchmark economy.

In other complementary work, Cordoba and Ripoll (2013) abstract from within-country heterogeneity in a model with credit frictions and find that fertility and mortality differences play important roles in explaining the cross-country dispersion of schooling attainment. While the model does well in explaining the overall world distribution of average schooling attainment, the dynamic nature of child development is not considered and, without heterogeneous agents, the model is unable to produce within-country educational inequality. Finally, Manuelli and Seshadri (2014) consider a production technology for human capital

²As discussed in a briefing by James Heckman (2006), a natural extension of his model of skill formation would be to include child health stocks. Heckman notes that literature on the economics of child development and the economics of health have largely grown in isolation from each other. He argues that augmenting his proposed technology of skill formation with health stocks would effectively create a model of “capacity” formation that could unite the two fields. This chapter essentially takes a step in that direction.

specific to early childhood and find support for cross-country differences in human capital stocks by age six due to the dynamic complementarity of investments. However, the model abstracts from credit frictions and within-country heterogeneity and is unable to account for the observed cross-country dispersion of average schooling attainment.³

The remainder of this chapter is presented as follows. Section 1.2 begins by providing some additional discussion and evidence linking education and early health. Section 1.3 builds the economic framework of the model, and Section 1.4 describes the calibration strategy. Section 1.5 provides the baseline results of the quantitative analysis, while Section 1.6 presents a series of counterfactual exercises. Finally, Section 1.7 concludes.

1.2. EDUCATION AND EARLY CHILDHOOD HEALTH

There is a growing body of evidence that early childhood health can have a significant effect on future life outcomes, including cognitive development and investments in education (Yamauchi 2008; Currie and Almond 2011; Todd and Winters 2011; Currie and Vogl 2013). Perhaps the most convincing evidence stems from a number of experimental studies showing that childhood health interventions can have a direct causal effect on educational outcomes in the developing world. For example, several randomized trials found that nutritional supplement programs had large impacts on test scores and schooling attainment for children in Guatemala (Pollitt et al. 1993; Maluccio et al. 2009) and Jamaica (Grantham-McGregor et al. 1991). In another well known study, Miguel and Kremer (2004) show that a deworming program in Kenya greatly increased school attendance. Overall, there is strong micro-level support for a mechanism in which early childhood health has a meaningful impact on later educational investment decisions.

In addition to the empirical micro evidence linking early health to education, Table 1.1

³See Cordoba and Ripoll (2013) for a series of experiments showing this inability.

provides some correlations at the country-level to gauge if my mechanism is consistent with patterns observed in aggregated data. Specifically, results are shown for a number of cross-country regressions on average years of schooling for 25-29 year olds.⁴ These results should be viewed as simple correlational relationships that serve to motivate my modeling choices and provide support for the mechanism driving the results of my quantitative analysis. The results displayed in column (1) reconfirm the positive relationship between schooling attainment and national income levels shown in Figure 1.1. The remaining specifications explore the relationship between schooling attainment, financial development, early health, and inequality.

Table 1.1: Dependent Variable: Average Years of Schooling (age 25-29)

| | (1) | (2) | (3) | (4) |
|-----------------------|---------------------|---------------------|----------------------|----------------------|
| <i>ln GDP</i> | 2.050*** (0.258) | 1.554** (0.278) | 0.936** (0.294) | 0.616** (0.213) |
| <i>FD</i> | | 0.016*** (0.004) | 0.003 (0.005) | 0.002 (0.004) |
| <i>Stunting</i> | | | -0.062*** (0.019) | -0.040*** (0.014) |
| <i>Wasting</i> | | | -0.079* (0.041) | -0.002 (0.031) |
| <i>Sch Gini</i> | | | | -9.474*** (1.082) |
| <i>Obs</i> | 91 | 91 | 91 | 91 |
| <i>R</i> ² | 0.869 | 0.887 | 0.908 | 0.953 |

Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Observations weighted by country population. Includes year and region dummies.

As credit frictions play an important role in my theory, I first examine the relationship between average schooling attainment and a country's level of financial development (*FD*). Here I follow the financial development literature by using data from the World Bank on domestic credit to the private sector as a percentage of GDP as a proxy for credit market efficiency. Column (2) shows that the average years of schooling in the young adult pop-

⁴Schooling data is from Barro and Lee (2010) for 2005 if available, else 2000.

ulation is positively associated with the financial development proxy. This provides some preliminary evidence that deeper financial markets may help alleviate the extent to which borrowing constraints bind, allowing higher average investment in human capital over the life-cycle.

Next I attempt to more directly explore the relationship between cross-country measures of early childhood health and average schooling attainment. However, comprehensive measures of health are not available across countries; therefore, as a proxy I use World Bank data on stunting and wasting prevalence in children under five.⁵ Column (3) adds these variables to the previous specification. Quantitatively speaking, a one standard deviation decrease in the prevalence of stunting (15.9%) or wasting (5.2%) is associated with an increase in average schooling of 1.0 or 0.4 years—both statistically significant relationships. Note that these measures are somewhat ambiguous in terms of how they relate to overall health distributions within a country. They are most likely capturing a combination of average early health stocks and health inequalities within the country. In either case, I view these relationships as consistent with a mechanism in which poor early health results in less schooling over childhood. Note also that the coefficient on financial development falls to an insignificant level after adding the health measures. This suggests that early health may be a relevant channel through which credit frictions affect educational investment decisions.

Finally column (4) adds a schooling Gini coefficient to the regression.⁶ First note that there is a strong negative relationship between schooling inequality and average attainment even after controlling for income levels.⁷ The relationship is also quantitatively meaningful—a one standard deviation reduction in the schooling Gini (0.18) is associated with an increase in average schooling of 1.7 years. Second notice that the estimated coefficient on wasting

⁵Prevalence of child stunting is the percentage of children under age 5 whose height-for-age is more than two standard deviations below the median for the international reference population ages 0-59 months. Wasting is defined analogously using weight-for-height.

⁶Schooling Gini coefficient calculated for each country with procedure proposed by Castello and Domenech (2002). Similar to an income Gini, the coefficient takes values from 0 to 1, with a higher value reflecting more inequality in schooling attainment.

⁷For further evidence, see Thomas et al. 2002; Castello and Domenech 2002; Castello-Climent 2010.

prevalence falls to near zero while that on stunting falls by roughly 65%. This is generally consistent with the mechanism examined in my model. Namely, the effects of early health inequalities on average schooling attainment can be captured through their effects on schooling inequality.

1.3. ECONOMIC FRAMEWORK

Consider a world in which each country is a closed economy populated by a large number of households that each consist of overlapping generations of a family. Family members are altruistic towards each other and make decisions as a single economic unit. Over time, children in the family grow up, have children of their own, and eventually replace their parents in the household. In this way each household in the economy is an infinitely lived dynasty. As children grow up, they accumulate human capital through both early health and later educational investments.

The key feature of the model is that investments take place over multiple periods and that individuals cannot fully borrow across periods to finance additional investments. This allows the model to capture the important interaction between borrowing constraints and the dynamic complementarity of human capital investments early in life. My quantitative analysis will focus on stationary equilibrium in each economy where prices and the aggregate behavior of households are constant over time. As such, I omit time subscripts from the description of the model and use only a prime superscript to denote variables one time period ahead.

1.3.1 Demographic Structure

Individuals live through four stages of life, in which they are referred to as a child (conception to age 6), youth (age 7-30), adult (age 31-54), and elder (age 55-78), respectively. Early

health investments are made during childhood, which is assumed to last from conception until formal schooling begins. As a youth, an individual goes to school then eventually begins working in the labor market. Also, near the end of the stage, a youth conceives a child of their own (at real age 23). As an adult and an elder, an individual simply works and eventually retires. The full life-cycle of an individual is shown in the first row of Figure 1.2. The last column shows members of a household at any given point in time.

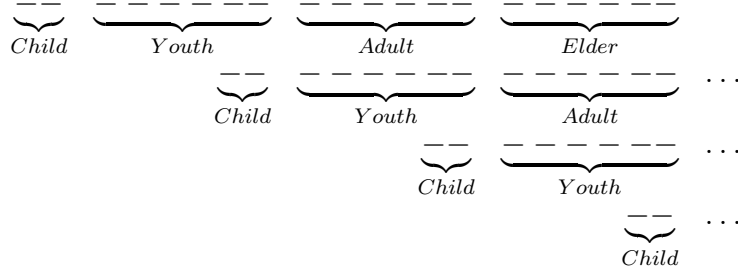


Figure 1.2: Generations of a Dynasty

1.3.2 Final Good Production

Households are assumed to derive utility from the consumption of a single final good. Aggregate output of the final good Y is assumed to be produced by a representative firm using the technology:

$$Y = AK^\alpha H^{1-\alpha} \quad \alpha \in (0, 1), \quad (1.1)$$

where K and H are the aggregate physical and human capital used in the final goods sector, and A is total factor productivity (TFP). Final goods can be consumed (C), invested in physical capital (I_K), or invested in human capital (I_H):

$$Y = C + I_K + I_H.$$

Finally, letting δ equal per-period depreciation, the law of motion of physical capital is given by:

$$K' = (1 - \delta) K + I_K.$$

1.3.3 Human Capital Production

Most cross-country models of human capital investments have relied on the simplifying assumption that time spent as a child can be represented as a single period. This implicitly assumes that inputs in the production of human capital are perfect substitutes over all stages of childhood. In contrast, recent evidence suggests that human capital accumulation is a dynamic process that begins early in life and interacts over multiple stages.⁸ In line with this view, I use a production framework that allows health investments made during early childhood to augment and affect the productivity of investments at later ages. Specifically, human capital is accumulated through investments in early childhood health ($i > 0$) followed by later investments in education ($e \geq 0$). Together, these inputs produce human capital according to the following production function:

$$h = [\pi i^\nu + (1 - \pi)(\lambda + e)^\nu]^\frac{1}{\nu},$$

where $\nu \leq 1$, $\pi \in [0, 1]$, $\lambda > 0$.

The parameter π is what Cunha and Heckman (2007) call a “skill multiplier,” which reflects the impact early investments have on human capital accumulation by both augmenting and increasing productivity of later investments. The parameter ν governs the degree of dynamic complementarity of investments, or how easy it is to make up for low investments in one period with investments in the other. Finally, the parameter λ allows some return to

⁸See Cunha et al. (2006) for an extensive review of the empirical literature and theoretic foundations for modeling human capital skill formation as a multi-stage process. See Cunha (2005); Todd and Wolpin (2007); Del Boca et al. (2014) for additional evidence.

early health investments even if a youth invests little or nothing in education. This is a key parameter for predicting schooling in developing economies where often non-trivial portions of the population never go to school or dropout very early.

Childhood health investments are modeled along two related dimensions—nutritional intake and medical care. This is the most common broad division in the health economics literature and is also convenient for later calibration of public subsidies and relative prices due to how available cross-country data is aggregated. Formally, composite early health investments are produced with inputs of food (d) and medical care (m) according to:

$$i = d^\chi m^{1-\chi}, \quad \chi \in [0, 1].$$

As a youth, educational investments are made through a combination of schooling time (s) and expenditures on educational quality (q) according to:

$$e = \theta s^\eta q^{1-\eta} \quad \eta \in [0, 1],$$

where θ is an idiosyncratic parameter that reflects a youth's learning ability. Learning ability is assumed to be correlated from one generation to the next through a simple Markov process.

The choice variable s takes values between zero and one and reflects the fraction of an individual's youth that is spent in school. Expenditures on educational quality are assumed to be a composite of human capital services (\tilde{h}) and educational goods (g):

$$q = \tilde{h}^\phi g^{1-\phi} \quad \phi \in [0, 1].$$

This captures the notion that both physical goods (e.g. books, buildings, transport) as well as human capital—primarily in the form of teachers—are necessary to realize meaningful returns from dedicating time to school.

1.3.4 Prices

Markets are assumed competitive and the representative firm in the final good sector chooses capital stocks to maximize profits. This implies that physical and human capital earn their marginal products:

$$w = (1 - \alpha) AK^\alpha H^{-\alpha} \quad (1.2)$$

$$r = A\alpha K^{\alpha-1} H^{1-\alpha} - \delta \quad (1.3)$$

where w is per unit wage rate for human capital, r is the interest rate on physical capital, and the price of the final good is set to one (the numeraire).

The relative prices of human capital investment goods in terms of the final good (P_f, P_m, P_g) are exogenously set during calibration. Given the relative price of food P_d and medical care P_m , the price of composite early health investments can be determined by a household static cost minimization problem:

$$P_i i = \min_{d,m} P_d d + P_m m$$

$$s.t. \ i = d^\chi m^{1-\chi}.$$

Setting $\hat{\chi} \equiv \frac{1-\chi}{\chi}$, the optimal choices are

$$m = \left(\hat{\chi} \frac{P_d}{P_m} \right)^\chi i, \quad d = \left(\hat{\chi} \frac{P_d}{P_m} \right)^{\chi-1} i.$$

This implies

$$P_i = \frac{\hat{\chi}^\chi}{1 - \chi} P_d^\chi P_m^{1-\chi}.$$

Notice that the price of composite early health inputs is increasing in both the price of food and medical care.

Similarly, given the relative price for inputs \tilde{h} and g , the price of composite investments

in educational quality can be determined by the static cost minimization problem:

$$P_q q = \min_{\tilde{h}, g} w \tilde{h} + P_g g$$

$$s.t. \ q = \tilde{h}^\phi g^{1-\phi}.$$

Setting $\hat{\phi} \equiv \frac{1-\phi}{\phi}$, the optimal choices are

$$g = \left(\hat{\phi} \frac{w}{P_g} \right)^\phi q, \quad \tilde{h} = \left(\hat{\phi} \frac{w}{P_g} \right)^{\phi-1} q.$$

This implies

$$P_q = \frac{\hat{\phi}^\phi}{1-\phi} w^\phi P_g^{1-\phi}.$$

The price of composite educational quality inputs is increasing in the price of human capital services (w) and educational goods (P_g).

1.3.5 Public Subsidies

A theory of education and health investments cannot abstract from the significant role of the public sector. In order to account for this on the education side, I model public expenditures as a subsidy to composite units of educational quality q . Specifically, expenditures on educational quality are subsidized at the rate p_q so that private expenditures equal $(P_q - p_q) q$. Because data used later to calibrate the model is only available for total public spending on education, the subsidy is assumed to be on the composite input q and not directly on inputs of human capital services \tilde{h} or educational goods g . This is equivalent to assuming that public funds are divided optimally between the two inputs. Public education subsidies are financed with a proportional tax on income τ_q .

Public expenditures on child health are modeled as a subsidy to child medical care m . Specifically, child medical care is subsidized at the rate p_m so that private expenditures equal $(P_m - p_m) m$. Public medical care subsidies are also financed with a proportional tax

on income τ_m , such that the total tax rate is given by: $\tau = \tau_m + \tau_q$. As there is no comparable cross-country measure of public spending on food subsidization, I do not explicitly include food subsidies in model. Note, however, that governments in many developing countries primarily effect food prices on the production side by subsidizing agricultural industries. Because in my quantitative analysis I adjust P_d across countries based on World Bank data on observed food prices (see Section 1.4.2), these subsidies would in effect be included in the adjusted prices. However, this adjustment would not require countries to pay for such subsidies in the model because they do not show up in the government budget constraint. Instead, these subsidies effectively show up in the calibrated model as exogenous productivity gains in the agriculture sector. In short, as long as these agricultural subsidies serve as a relatively small distortion through taxes, this distinction can be safely ignored.

1.3.6 Earnings and Asset Market

As a youth, an individual begins earning income in the labor market after they have completed school. As such, the total earnings for a youth is given by:

$$w\psi h(1-s),$$

where ψ is a life-cycle productivity parameter. As an adult, an individual's time is fully dedicated to work and earnings are given by:

$$w(h+z),$$

where z is an *iid* stochastic non-negative productivity shock. The productivity shock provides an important source of heterogeneity in the model and is unknown and uninsurable prior to an individual reaching adulthood. The shock is assumed to remain throughout the remainder

of an individual's life resulting in elder earnings given by:

$$\kappa w(h + z),$$

where κ is the portion of elderhood that is spent working prior to retirement. Finally, individuals are able to save or borrow a single risk-free asset (a) which earns the gross after-tax return R . For ease of future notation, denote the sum of adult and the present value of elder after-tax earnings as:

$$W(h, z) = (1 - \tau) [1 + R^{-1}\kappa] w(h + z).$$

1.3.7 Borrowing Constraints

The dynamic nature of child development implies that investments should optimally be spread out over time in a manner that takes advantage of complementarities. It is well known that this leaves scope for credit constraints during childhood to inhibit efficient investments in human capital (Cunha and Heckman 2007; Caucutt and Lochner 2012). Despite this and the evidence that early health matters for adult outcomes, no research has examined if borrowing constraints inhibit early health investments. However, there is a growing body of evidence that suggests the general effects of borrowing constraints may be most important at young ages.⁹ By explicitly modeling early childhood separately from schooling ages, my theory allows credit frictions to interact with investments over the dynamic process of child development. This implies that borrowing constraints may be an important source of cross-country schooling differences not only because they directly limit investments in education, but also because they result in underinvestment in early childhood health which lowers the returns of later going to school.

⁹For example, several studies have concluded that family income received early in life has a larger impact on educational outcomes than income received later in life (Duncan and Brooks-Gunn 1997; Duncan et al. 1998; Levy and Duncan 1999; Caucutt and Lochner 2006, 2012). Other studies find highest returns for disadvantaged children in early childhood programs (Blau and Currie 2006; Cunha et al. 2006).

As discussed in Lochner and Monge-Naranjo (2011), availability of loans is often explicitly linked to individual levels of human capital. The basic reasoning is that higher skilled individuals have higher expected future earnings and are thus able to credibly commit to repay more debt. This reasoning also implies that economies with stronger mechanisms for enforcing loan contracts (e.g. wage garnishments or exclusion from financial markets) will also experience less credit market inefficiencies. To capture these important credit market features, I employ endogenous borrowing constraints. Specifically, youths are able to borrow up to a fraction γ of the discounted value of their lowest possible earnings stream. Formally, this borrowing constraint is given by:

$$B(h) = \gamma R^{-1} W(h, \underline{z})$$

where \underline{z} represents the lowest possible productivity shock. The parameter γ thus reflects the efficiency of credit markets, or alternatively the relevant financial development of the economy. This may be of particular importance for families in developing economies where financial markets are often severely underdeveloped, thus limiting access to credit at early and late stages of life. Importantly, I allow borrowing limits to vary across countries according to the domestic level of financial development.

1.3.8 Decision Problem

Without loss of generality, the youth is assumed to make all decisions for the household given three state variables—household wealth (b), health investments made in the youth as a child (i), and the youth’s learning ability (θ). Given this state vector, a youth maximizes the present utility of the household plus the expected discounted utility of all future generations of the family dynasty. Specifically, a youth chooses household consumption c , assets a' , their own educational investments (s, q) and health investments in their young child i' , who is conceived towards the end of youth.

Using dynamic programming language, the decision problem faced by a youth can be written:

$$V(b, i, \theta) = \max_{c, s, q, i', a'} \{u(c) + \beta E_{z', \theta'} [V(b', i', \theta')]\}$$

subject to

$$\begin{aligned} c &= b + (1 - \tau) w \psi h (1 - s) - (P_q - p_q) q - P_i i' - a' \\ b' &= R a' + W(h, z') \\ h &= f(i, s, q, \theta), \quad a' \geq -B(h) \\ s &\in [0, 1], \quad c, q, i' \geq 0 \end{aligned}$$

where $u(\cdot)$ is current period utility and value function $V(\cdot)$ is the total expected discounted utility of arriving in a period of time with a given state vector. The first constraint is the household budget constraint equating consumption to household wealth plus youth earnings less expenditures on human capital and savings. The total cost of educational investments include subsidized expenditures on educational quality $(P_q - p_q)q$ as well as the forgone earnings from time spent in school. The second constraint defines household wealth the following period when the youth becomes an adult themselves. This wealth includes the gross return to household asset holdings as well as the youth's earnings as an adult.¹⁰

Note that any assets borrowed against the youth's future earnings (i.e. $a' < 0$) can be used on current household consumption c , educational investments in the youth (s, q) , or early health investment in the youth's child i' . However, due to the endogeneity of the borrowing constraint, if youths are not investing in their own human capital, their borrowing capability will be limited. Note also that the value function next period is only known in expectation due to the stochastic nature of future productivity and ability shocks. This implies that while a

¹⁰Adults are not restricted from fully borrowing against their elder earnings so the present value of elder earnings can simply be included as part of adult earnings. Any earnings of the youth's parent as an elder are thus implicitly included in asset holdings a' .

youth knows their learning ability prior to making educational investments, they do not know the productivity shock they will receive as an adult. Furthermore, the youth must make early health investments in their own child without knowing the child's learning ability. Recall that due to the dynamic nature of human capital accumulation, early health investments are potentially important when choosing later levels of educational quality expenditures and schooling.

1.3.9 Definition of Equilibrium

A stationary competitive equilibrium is a set of value functions, decision rules, prices, subsidy rates and an invariant distribution of households across states, such that (i) given prices, subsidies, and the tax rate, households solve their optimization problem; (ii) given prices, firms maximize profits; (iii) physical and human capital markets clear and the aggregate resource constraint holds; (iv) government budget is balanced. For a detailed definition of the equilibrium in the model economy, see Appendix A.

1.3.10 Sources of Heterogeneity

Before moving on to the empirical calibration strategy, it is useful to summarize the sources of heterogeneity in model (see Table 1.2). Differences in family wealth and individual learning ability drive heterogeneity in human capital investments within a country. However, differences in the stationary distribution of family wealth and human capital in each economy is determined by heterogeneity at the country level. In this chapter, I focus on cross-country differences along three dimensions—prices, public subsidies, and financial development. Price heterogeneity is driven by productivity differences in the production of final and investment goods. Productivity remains the most common means of explaining cross-country distributions of national income and other outcomes in the macroeconomic literature. Public subsidies to early health and education are substantial in most countries of the world and may help increase or redistribute investments across the household population. Finally, fi-

nancial development governs how much parents can borrow against future earnings to invest in themselves or their children. The extent to which financial constraints restrict human capital investments determines how closely health and education is tied to family wealth within a country. In the next section I discuss in more detail how each of these features are calibrated across countries using empirical data.

Table 1.2: Sources of Heterogeneity

| | Within-Country | Cross-Country |
|-----------------------|----------------|-----------------------|
| Ability | θ | - |
| Productivity | z | - |
| Prices | - | w, R, P_d, P_m, P_g |
| Public subsidies | - | p_m, p_q |
| Financial Development | - | γ |

1.4. CALIBRATION

I use a calibrated version of the model to quantitatively assess the impact of early health differences on educational outcomes among a cross-section of countries. Due to data availability, calibration and later counterfactual experiments are conducted on a sample of 71 countries. There is little empirical evidence or theoretical precedence to directly pin down many parameters in the model. However, I take the stance that certain processes such as human capital development and the inter-generational transmission of ability are universal in nature. As such, I take advantage of the availability of rich longitudinal and cross-sectional data in the U.S. to restrict the parameters governing these processes. Specifically, universal parameters are calibrated so that the baseline equilibrium of the U.S. model economy matches a number of relevant moments from the U.S. data. Other parameters that govern heterogeneity across countries such as prices and public subsidies are calibrated for each country individually. This section describes these calibrations in turn.

1.4.1 Parameters Common Across Countries

Parameters governing preferences, production technologies (except TFP), life-cycle productivity, and random processes are assumed to be common across all countries. For ease of exposition, I will discuss calibration for each of these groups in turn, but to be clear, all U.S. data moments were targeted simultaneously in order to pin down common parameters.

1.4.1.1 Preferences and Final Good Production The household utility function is assumed to be given by:

$$u(c) = \frac{c^{1-\sigma}}{1-\sigma}$$

where $\sigma \geq 0$. I set the relative risk aversion parameter $\sigma = 2$ while the discount factor β is set to target an annual after-tax interest rate in the U.S. of 5% (Browning et al. 1999). For final good production, I set the capital share parameter $\alpha = 0.33$ which is consistent with the capital-income share in the U.S. (Gollin 2002). Finally, the annual depreciation rate is most commonly set in the 5-10% range in the macro literature, as such δ is set to an annual rate of 7.5%. Table 1.3 gives the set of preference and final good production parameters that are common across all countries.

Table 1.3: Preferences and Final Good Production Parameters

| Parameter | Value | |
|---------------------|----------|------|
| CRRA | σ | 2 |
| Discount factor | β | 0.28 |
| Capital share | α | 0.33 |
| Period depreciation | δ | 1.8 |

1.4.1.2 Human Capital Production Since my theory is primarily concerned with human capital investment decisions, it is important to carefully consider how human capital production parameters are selected. The six parameters governing the production of human

capital $\{\eta, \nu, \pi, \lambda, \phi, \chi\}$ are calibrated to match the following six targets for the U.S.:

1. Average years of schooling of 13.4, computed from CPS data on 30 year olds in 2005.
2. Correlation of schooling attainment to conditional parental earnings from conception to age six of 0.18. This correlation is calculated after conditioning on parental earnings from ages 7 to 30. This statistic is computed using data from the National Longitudinal Survey of Youth (NLSY) for the 1979 parental cohort.¹¹
3. Average annual private expenditures on child health from age 0-5 of 4.5% of per capita GDP for the middle tercile of parental income. To remain consistent with the model, this statistic is computed by combining expenditure estimates for food and health care reported by the U.S. Department of Agriculture (USDA) for 2005 (Lino 2006).¹²
4. High school dropout rate of 0.12, computed from CPS data on 30 year olds in 2005.
5. Teacher and staff compensation share of total educational expenditures of 0.67 for 2005, computed from U.S. Department of Education, Digest of Education Statistics (2008).
6. Ratio of annual private food to health care expenditures on children age 0-5 of 0.5, computed from USDA data for 2005.

Though the parameters interact to affect the targeted moments in complex ways, each parameter has meaningful effects on specific targets. The teacher salary share of educational quality inputs ϕ is directly pinned down by teacher and staff compensation share of total educational expenditures. Likewise, the ratio of annual private food to health care expenditures on children aged 0-5 is used to directly pin down the food share of early health investments

¹¹Parental earnings are calculated from reported family income between \$200 and \$275,000 averaged over the specified ages of the child. All earnings are adjusted to 2005 dollars using the CPI-U and discounted to the child's birth year using a 5% interest rate.

¹²Does not include health care expenses covered by insurance. However, I use out-of-pocket spending to set public medical care subsidies. This is equivalent to including employer provided insurance as part of public expenditures on health which is funded with taxes on income.

χ . Given the other parameters, average years of schooling sharply increases with the schooling time share η while average private health spending on children is increasing in the skill multiplier π . Recall that the parameter λ determines how productive a youth can be in the labor market without making educational investments. As a result, this parameter plays a primary role in determining the opportunity costs of attending school during the early ages of youth. As the youth continues to attend school, the importance of this parameter diminishes quickly. As such, the high school dropout rate is quite sensitive to increases in λ .

Finally, the complementarity between early health and later educational investments ν targets the conditional correlation of schooling attainment to annual parental earnings from conception to age six. Identification of this parameter relies on the fact that some children receive fewer health investments because their parents are financially constrained, but the family receives a high income shock when the child becomes a youth. The degree of complementarity will limit how much such youths will invest in education relative to a youth of equal wealth and ability but more early health investments.

Table 1.4 shows the calibrated parameters and a comparison between the data targets and the simulated moments from the U.S. benchmark economy. The calibration matches the targeted moments very well. Notice that the calibration results in strong dynamic complementarity of investments: $\nu = -1.3$ implies an elasticity of substitution between health and education investments of 0.43. Although there is no direct empirical comparison for this measure, it is quite similar to the elasticity estimates over childhood reported by Cunha et al. (2010) and Caucutt and Lochner (2012).

1.4.1.3 Life-cycle and Random Processes Recall that within country heterogeneity comes from idiosyncratic learning ability and productivity shocks. For households in all countries, ability is assumed to follow an AR(1) process in logs:

$$\ln(\theta') = \rho \ln(\theta) + \epsilon_\theta, \quad \epsilon_\theta \sim N(0, \sigma_\theta^2).$$

Table 1.4: Human Capital Production Parameters

| Parameter | | Value | Target | Data | B.E. |
|-------------------|-----------|-------|--|------|------|
| School time share | η | 0.46 | Mean years of schooling | 13.4 | 13.4 |
| Complementarity | ν | -1.3 | Corr of schooling to parent earnings (age 0-6) | 0.18 | 0.18 |
| Skill multiplier | π | 0.05 | Private health expend. (age 0-5-% GDP p.c.) | 4.5 | 4.5 |
| Base productivity | λ | 0.16 | High school dropout rate | 0.12 | 0.12 |
| Teacher share | ϕ | 0.60 | Salary share of educational expenditures | 0.67 | 0.67 |
| Food share | χ | 0.67 | Food to health care expend. ratio (age 0-5) | 0.5 | 0.5 |

I approximate this process over seven discrete values for ability using the Tauchen (1986) method. Productivity shocks are assumed to be *iid* log normal:

$$\ln(z) \sim N(\mu_z, \sigma_z^2),$$

and are similarly approximated over ten discrete values.

In addition to parameters governing the random processes, there are two remaining life-cycle parameters — ψ which governs youth productivity, and κ which adjusts elder earnings for retirement. The retirement parameter κ is set to a retirement age of 65. The five remaining parameters $\{\psi, \mu_z, \sigma_z, \sigma_\theta, \rho\}$ are calibrated to match the following five targets for the U.S.:

1. College graduation rate of 0.32, computed from CPS data on 30 year olds in 2005.
2. Average adult earnings to GDP per capita ratio of 1.4, computed from CPS data in 2005.¹³
3. Standard deviation of log of permanent earnings of 0.7 (Kopczuk et al. 2010).
4. Standard deviation of schooling of 2.9, computed from CPS data on 30 year olds in 2005.
5. Inter-generational correlation of schooling of 0.46 (Hertz et al. 2007).

¹³Earnings calculated using full-time year-round male workers aged 31-54 and are discounted to age 42 using a 5% interest rate.

While λ plays an important role in dropout decisions during early years of schooling, youth productivity ψ has a more pronounced effect at the top end of the schooling distribution. As such, this parameter effectively targets the college graduation rate. The mean and standard deviation parameters for the productivity shock (μ_z, σ_z) target the mean and standard deviation of earnings of adults. Finally, given the other parameters, the standard deviation and persistence of the ability shock (σ_θ, ρ) have strong effects on the standard deviation and intergenerational correlation of schooling. Table 1.5 shows the calibrated parameters and a comparison between the data targets and the simulated moments from the benchmark economy. Again, the calibration matches the targeted moments quite well.

Table 1.5: Life-cycle and Random Processes Parameters

| Parameter | | Value | Target | Data | B.E. |
|-----------------------------|-----------------|-------|---------------------------|------|------|
| Youth productivity | ψ | 0.24 | College graduation rate | 0.32 | 0.35 |
| Retirement share | κ | 0.42 | Retirement age | 65 | 65 |
| Productivity shock log-mean | μ_z | -4.1 | Mean earnings to GDP p.c. | 1.4 | 1.4 |
| Productivity shock std | σ_z | 2.0 | Std of log earnings | 0.7 | 0.7 |
| Ability std | σ_θ | 0.43 | Std of schooling | 2.9 | 2.9 |
| Ability persistence | ρ | 0.44 | Correlation of schooling | 0.46 | 0.40 |

1.4.2 Country-specific Parameters

In addition to the universal parameters that are assumed to be common in all economies, a number of parameters are assumed to vary across countries. These country-specific parameters include prices, public subsidies to education and medical care, and financial development.

1.4.2.1 Prices Wages and interest rates are allowed to vary across countries not only due to differing levels of capital investments, but also due to differences in TFP. I allow for this

by rewriting equations (1.2) and (1.3) and calculating prices in the baseline economies as:

$$\begin{aligned} w &= \frac{(1 - \alpha) y_{data}}{\bar{h}} \\ r &= \alpha \frac{y_{data}}{\bar{k}} - \delta \end{aligned}$$

where y_{data} is PPP adjusted GDP per capita from the Penn World Tables averaged from 2001-2005, and \bar{h} and \bar{k} are the endogenous per capita stocks of human and physical capital.¹⁴ Essentially, final output is fixed to observed levels in the data and TFP is adjusted to ensure the aggregate resource constraint clears in equilibrium. Note that after the baseline stationary equilibrium is computed for any country, that country's TFP can be backed out by rewriting equation (1.1) :

$$A = YK^{-\alpha}H^{\alpha-1}.$$

This yields the TFP value that is fixed in later experiments, where new output levels are endogenously determined after a counterfactual change.

In the U.S., the relative prices of human capital investment goods in terms of the final good are normalized to one — $P_f^{US} = P_m^{US} = P_g^{US} = 1$. I further assume that the local cost of teachers (i.e. the endogenous wage rate) determines the cross-country difference in the price of composite educational quality inputs P_q , by setting the the relative price of educational goods to one for all countries ($P_g = 1$). However, for each country I adjust the prices of health inputs directly to account for relative productivity differences in the food and medical care sectors. Specifically, I use data from the World Bank's International Comparison Program (ICP) to adjust the relative price of health inputs (P_d, P_m) using the local price of food and medical services relative to GDP reported for 2005.

¹⁴In theory, output of the final good y should be calibrated using GDP data net of teacher services valued at international prices and \bar{h} should be per capita human capital net of teacher services \tilde{h} . However, since these values are relatively small and to avoid a number of issues surrounding the appropriate choice of an international wage of teachers (see Erosa et al. 2010 for a discussion), I abstract from these adjustments.

1.4.2.2 Public Subsidies The subsidy rate to early medical care expenditures p_m , is set to match out-of-pocket health care spending as a share of total health care spending reported by the World Bank.¹⁵ For example, the U.S. out-of-pocket cost is reported as 14% so the subsidy rate in the U.S. is set to $p_m^{US} = 0.86P_m^{US}$. There is substantial variation in out-of-pocket health care rates across countries, with many developing countries realizing significantly higher rates than richer countries (see Figure 1.3).

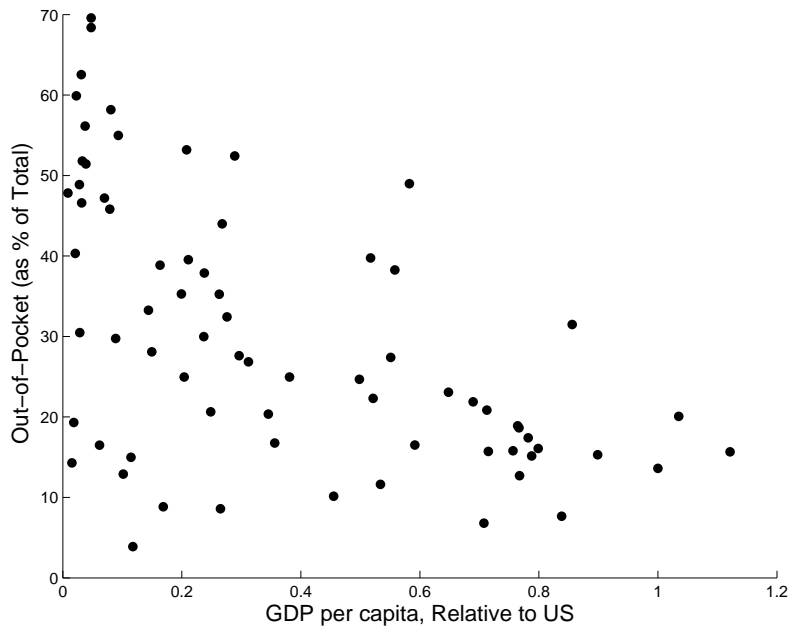


Figure 1.3: Health Care Costs. Source: World Development Indicators, The World Bank.

While health subsidies are directly pinned down by the data, baseline education subsidies are calibrated for each country during computation of the stationary equilibrium. Specifically, education subsidies p_q are set to target data on total public expenditures on education as a percentage of GDP from the Word Bank (see Figure 1.4). In a set of policy experiments, I later consider the implications of adjusting health versus educational subsidies in the developing world.

¹⁵Out-of-pocket spending is most consistent with the USDA data of early health spending used in the U.S. calibration, which does not include expenses paid by insurance.

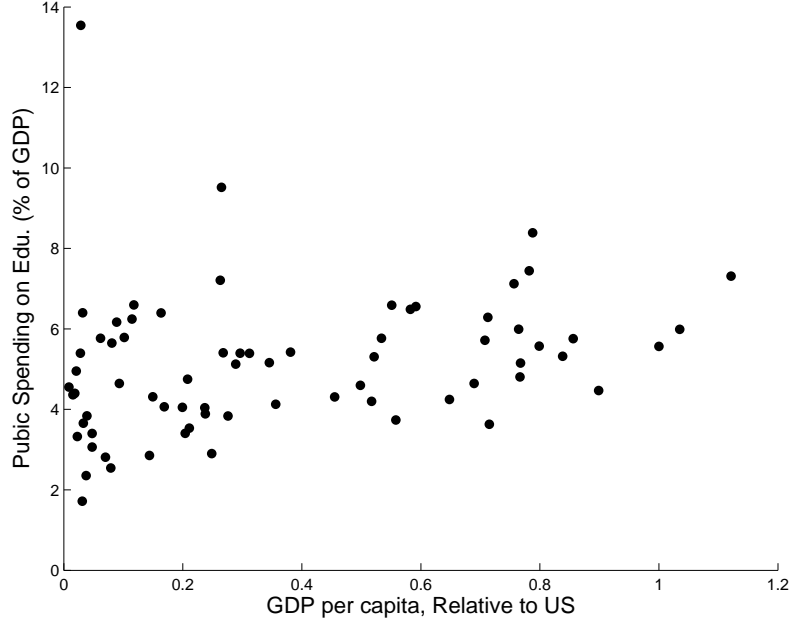


Figure 1.4: Public Spending on Education. Source: World Development Indicators, The World Bank.

1.4.2.3 Financial Development As there is no direct measure of the efficiency of credit markets as modeled in my theory, I use a slightly modified strategy to vary the financial development parameter γ across countries. First, I calibrate the parameter for the U.S.—again using a data moment to restrict the value. Second, I use a common financial proxy to adjust the parameter across countries. I briefly describe these two steps in turn.

Without credit frictions in my model, earnings would only be correlated across generations due to any persistence of learning ability. Thus, holding persistence and other parameters constant, the correlation of earnings is negatively related to financial development—given that at least some individuals face binding constraints. As such, I calibrate γ_{US} by targeting an inter-generational correlation of log-earnings of 0.44 as reported in Lee and Solon (2009). Restricting the financial development parameter in the U.S. to match the moment results in a calibrated value of $\gamma_{US} = 0.3$, implying that youths in the U.S. are able to borrow a maximum of 30% of the present value of their lowest possible adult earnings.

After calibrating γ_{US} for the benchmark economy, I use World Bank data on domestic credit to the private sector to proxy each countries relative level of financial development.

Formally, for any country j , the financial development parameter is calculated as:

$$\gamma_j = \frac{FD_j}{FD_{US}} \times \gamma_{US}$$

where FD is the domestic credit to the private sector as a percentage of GDP averaged from 2001-2005. In this way, I simply scale the financial development parameter of the U.S. economy by the relative domestic credit in each country. There is substantial variation across countries when using this measure of development (see Figure 1.5). I view this as a simple yet meaningful way to capture important heterogeneity across countries along this dimension.

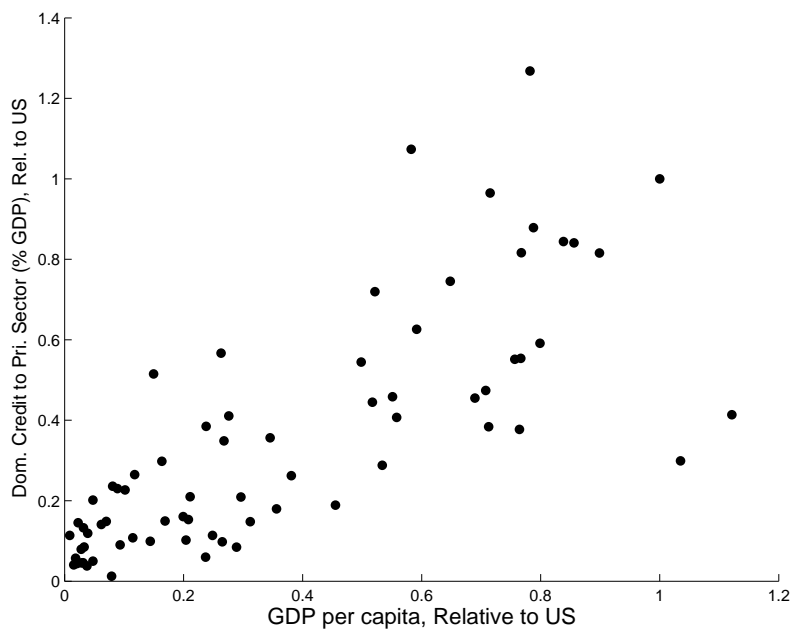


Figure 1.5: Financial Development. Source: World Development Indicators, The World Bank.

1.5. BASELINE RESULTS

This section begins by assessing the ability of the model to predict the cross-country dispersions of schooling attainment and schooling Gini coefficients observed in the data. Additional features of the baseline economies are then detailed. Throughout the quantitative analysis, I define developing countries as those with PPP adjusted GDP per capital under \$10,000.¹⁶

1.5.1 Fit of the Model

After computing the baseline equilibrium for the full set of 71 countries in the sample, I assess the fit of the calibrated model to observed schooling inequality within and across countries. The schooling attainment and schooling Gini coefficients from the data and the model are presented in Figures 1.6 and 1.7, while Table 1.6 displays a number of summary measures that are useful in assessing the overall performance of model. While calibrated to match schooling outcomes for the U.S. only, the model does quite well in predicting average attainment and inequality across the set of sample countries. The predicted cross-country mean of average schooling attainment is 10.5 in the model and 9.6 in the data while the predicted cross-country mean of the schooling Gini is 0.19 in model and 0.18 in the data.

Table 1.6: Model's Performance

| Measure | Cross-Country Mean | | Corr. | Cross-Country S.D. | |
|------------------------|--------------------|-------|-------|--------------------|-------|
| | Data | Model | | Data | Model |
| Ave Years of Schooling | 9.6 | 10.5 | 0.87 | 2.76 | 2.69 |
| Schooling Gini | 0.18 | 0.19 | 0.73 | 0.14 | 0.10 |

Although cross-country standard deviations were not targeted in calibration, the model also does well in predicting the dispersion of schooling measures across countries. Specifically, the model predicts 97% of the standard deviation of average attainment and 71% of the

¹⁶This is roughly consistent with the World Bank definition of developing countries in 2005. Out of the 71 sample countries, 33 fall in this category.

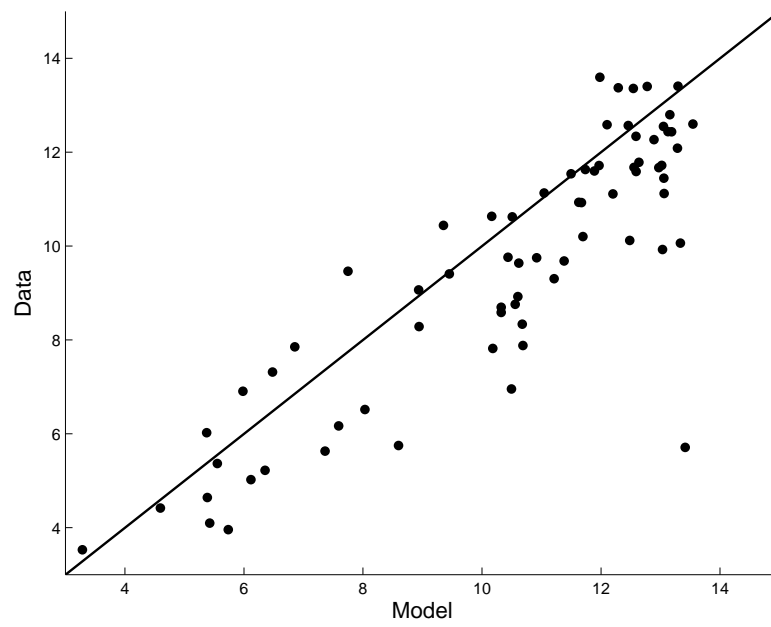


Figure 1.6: Average Schooling Attainment

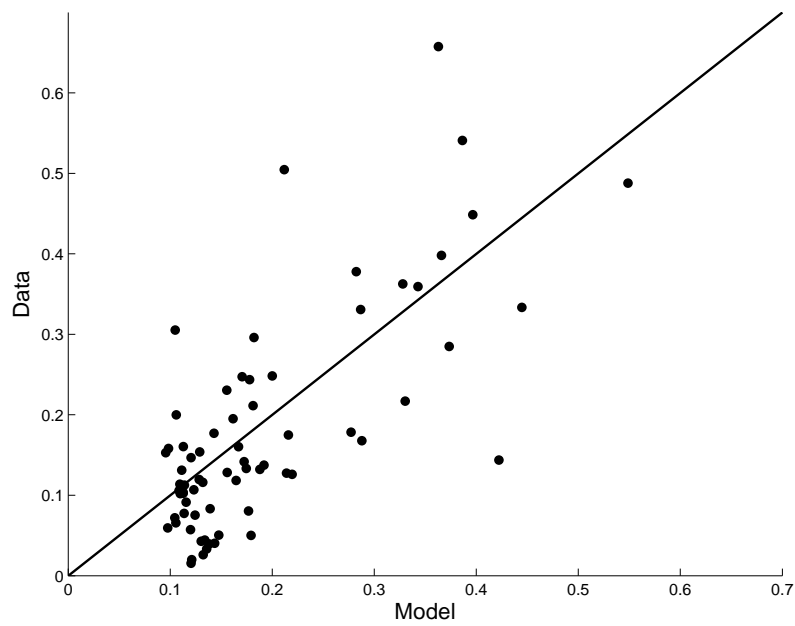


Figure 1.7: Schooling Gini Coefficients

dispersion of the schooling Gini. Furthermore, the correlation between schooling attainment in the model and the data is quite high at 0.87. The correlation is slightly lower for the schooling Gini at 0.73 as the model somewhat over-predicts the coefficient for high-income countries, where inequality is relatively low.¹⁷ Overall however, the model does quite well in explaining the patterns of schooling attainment and inequality observed across sample countries.

1.5.2 Additional Features of the Baseline Economies

The model predicts that human capital varies significantly more across countries than schooling attainment levels (see Figure 1.8). For example, the average human capital among developing countries is only 36% that of U.S. while schooling attainment is 61%. This suggests that standard human capital estimates based only on years of schooling would under-estimate differences between the U.S. and the developing world by almost 60%. The increased dispersion in human capital over schooling estimates is driven by differences in early childhood health and later educational quality investments—average investments in the developing world amount to only 18% and 16% of U.S. levels, respectively (see Figure 1.9).

In order to examine the mechanism linking early health to education it is also useful to examine how *within-country* inequality differs across countries. As national income rises, early health inequality falls sharply among the very poorest countries in the sample before leveling off and then slowly rising among high-income countries (see Figure 1.10).¹⁸ This pattern is consistent with very high returns up to some minimal amount of early health investment, at which point returns to additional investments are only achieved by accompa-

¹⁷Note however that comparing schooling Ginis from the model and data is non-trivial. While model calculations are based on a known continuous schooling variable for each individual, empirical schooling Ginis are calculated from aggregate estimates of discrete data. For example, empirical estimates use the fraction of individuals who reach certain discrete cut-points (e.g. the percentage who complete primary school) and estimates of average attainment at each discrete level (e.g. average years of primary schooling). Furthermore, the specific cut-points used in estimation vary from country to country (e.g. primary school ends at grade 6 or 8).

¹⁸For consistency with the measurement of schooling inequality, health inequality is also measured through a Gini coefficient.

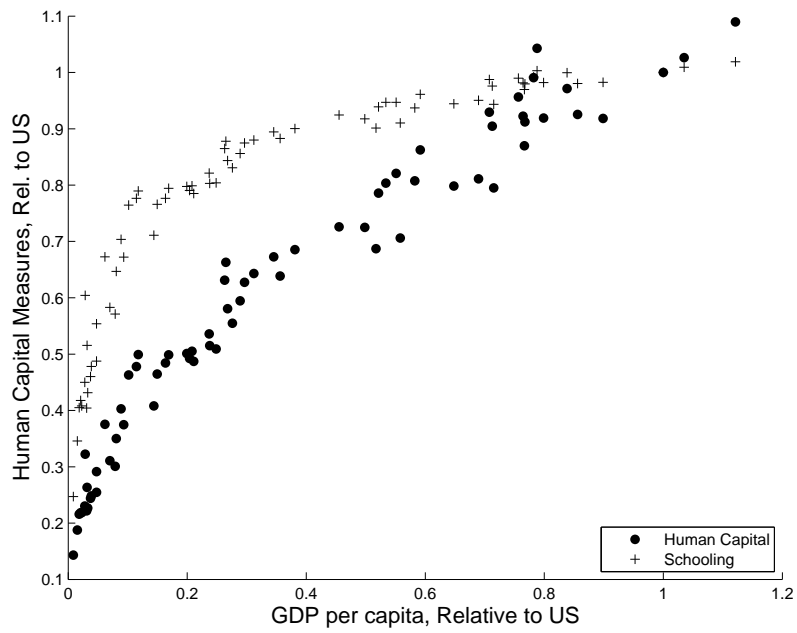


Figure 1.8: Human Capital and Schooling Attainment

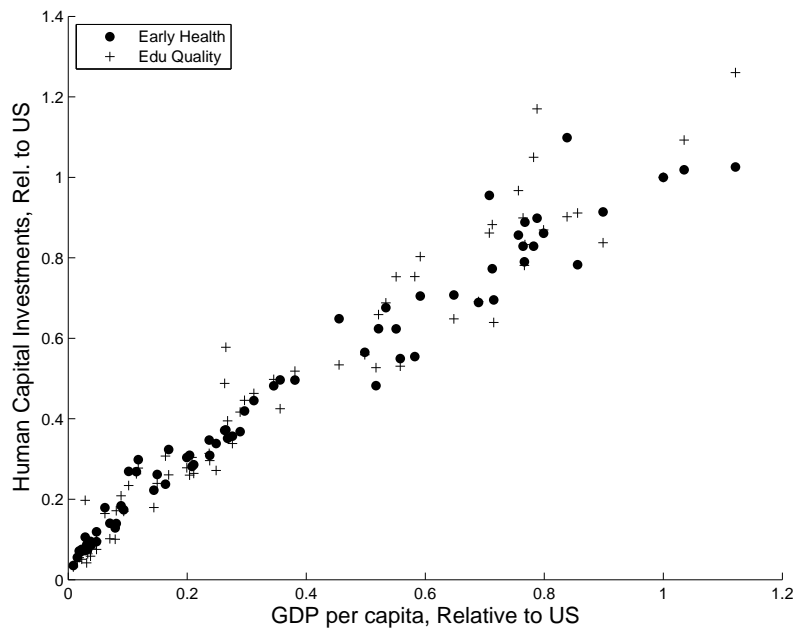


Figure 1.9: Health and Education Quality Investments

nying them with substantial educational quality inputs. When examining the relationship between early health inequality and later educational inequality, several relationships emerge. First, educational investment Ginis are significantly higher than early health Ginis across all countries in the sample (see Figure 1.11). Second, there is positive correlation between the two measures, predicting that countries with more early health inequality also experience more inequality in later educational investments. These observations are consistent with early health inequalities being amplified into even larger gaps in educational investments in the model. In the next section, I attempt to quantify this amplification mechanism using a number of counterfactual experiments.

Finally, there is substantial variation in the percentage of households in each country that borrow up to their limits in the baseline (see Figure 1.12). In the U.S., 14% of families are financially constrained compared to an average of 41% among developing countries. These measures are consistent with lower levels of financial development and human capital among developing countries—recall borrowing constraints are endogenously determined by an individual’s ability to repay.

In the U.S., it is relatively poor youths that receive a high ability shock that are borrowing constrained. Since all children in the U.S. receive a relatively substantial early health investment, even the poor youths have enough invested in their early health to make it worthwhile to borrow to take advantage of their high learning ability. All else equal, high ability parents also invest somewhat more in the health of their child than lower ability parents due to complementarities and the persistence of ability across generations. As a result, even though constrained youths choose marginally lower health investments in their children than unconstrained youths of similar ability, overall they actually invest 3% *more* than the average unconstrained parent. Compare this to the developing world where constrained parents invest about 23% less in their child’s health than unconstrained parents. Here it is poor youths with little invested in their early health that would like to borrow over their limits in order to finance additional consumption as well as investments in their child’s health.

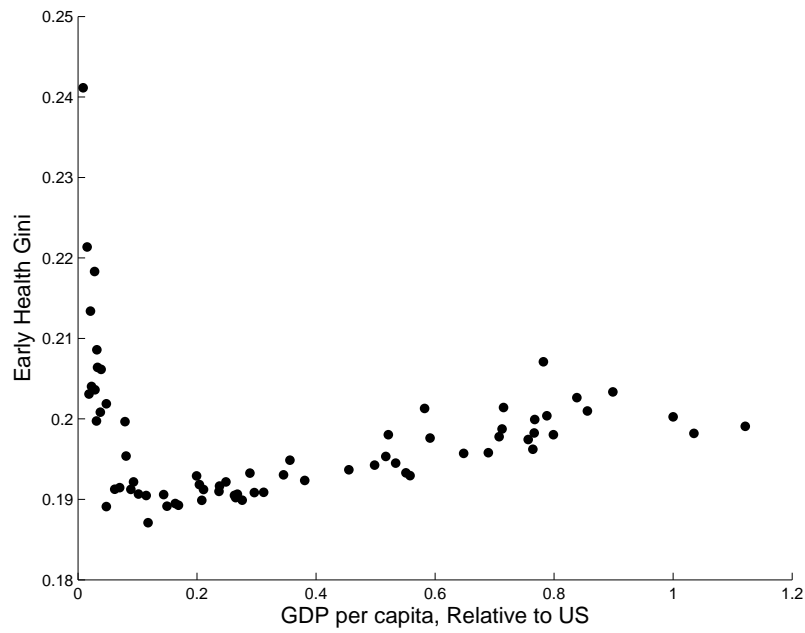


Figure 1.10: Early Health Inequality

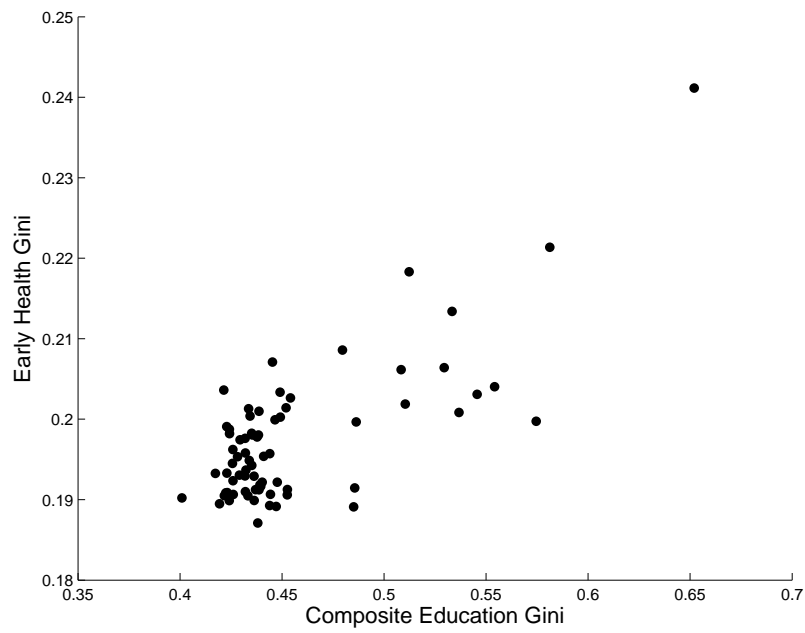


Figure 1.11: Early Health and Education Inequality

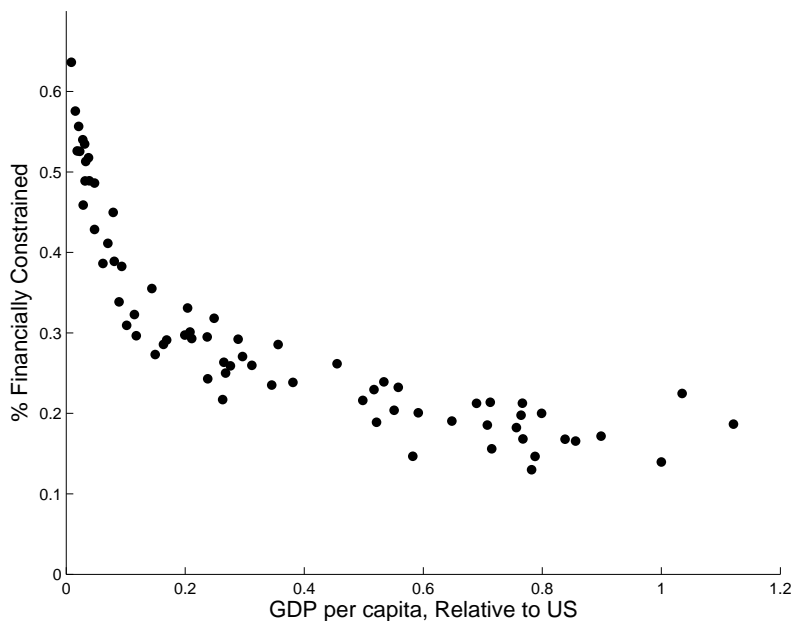


Figure 1.12: Financially Constrained

In other words—while credit frictions predominately limit educational investments made by some high ability youths in the U.S., in the developing world they also substantially limit early health investments of poor parents. These features of the baseline economies highlight the differing effects of credit frictions in determining human capital investments over alternate stages of child development.

1.6. COUNTERFACTUAL EXPERIMENTS

In this section I discuss a number of counterfactual experiments conducted on the same sample of 71 countries used in the baseline analysis. First I attempt to isolate and quantitatively assess the effects of early health inequalities on education and other outcomes. I do this by eliminating early health differences within each country, then across all countries. Lastly, I discuss a set of experiments that compare the effects of increasing early health versus educational subsidies in terms of efficiency and equity of investments.

1.6.1 Equating Early Health *Within* Countries

The strong complementarity between early health and educational investments identified in the baseline calibration suggests that early health inequalities play a potentially significant role in determining later educational inequalities. In order to quantitatively assess this mechanism, I simulate a counterfactual set of economies in which early health inequalities are eliminated. Specifically, in this experiment I equate i to the maximum baseline level *within* each country. In other words, for each country I fix early health investments for all children to the highest level that was realized by any child in that country in the baseline. I then allow taxes to adjust in order to pay for the policy change (while keeping the percentage of output devoted to public education τ_q at the baseline level). In practice, this is equivalent to a policy of universal food and medical care for young children at a fixed level where additional investments are not possible. Average percentage change from the baseline economies across the full sample and for the subset of developing countries are reported in Table 1.7 for selected outcomes.

Table 1.7: Equating Health *Within* Countries (Ave % Change)

| Countries | <i>School</i> | | $\log y$ | <i>Consumption</i> | | % <i>Constrained</i> |
|------------|---------------|-------------|----------|--------------------|-------------|----------------------|
| | <i>Ave</i> | <i>Gini</i> | | <i>Ave</i> | <i>Gini</i> | |
| All | 17.6 | -4.7 | 1.8 | 4.4 | -9.6 | -0.6 |
| Developing | 27.6 | -12.0 | 2.0 | 0.2 | -11.7 | -9.8 |

Raising investments to eliminate early health differences within countries reduces schooling Ginis by 4.7% and increases average schooling attainment by 17.6% on average across all countries. When focusing only on developing countries, these changes are significantly stronger—schooling Ginis fell by 12% and schooling attainment rose by 27.6% on average. These results occur because the increase in early health raises the productivity of later educational investments due to complementarities, particularly for the children of poor, financially constrained parents. This effect is then compounded over time as poor but healthier children ultimately realize higher earnings as adults and are able to transfer more resources to their

child who can then invest more in their own education. This quantitatively demonstrates my first main finding—early health inequalities in developing countries are amplified into larger schooling inequalities. This amplification effect is much smaller in higher income countries where fewer parents are constrained and most children are healthy enough in the baseline to realize gains from attending school for a significant length of time. In these countries, further increases in early health levels are accompanied by smaller changes in schooling due to the decreasing returns and increasing opportunity costs of attending school as well as more youths reaching their borrowing limit (see discussion below).

In addition to schooling attainment, output also rose on average as a result of the experiment. Despite this, consumption remained unchanged on average in developing countries where increased national income was used mostly to finance the new health policy as well as increased spending on education. However, consumption inequality between households—measured through a consumption Gini—fell by almost 12%. Thus, although there was no level effect on consumption in developing economies, there was a redistributive effect.

The experiment also results in two opposing effects on the borrowing choices of families. First, because health is fixed for all children, poor youths do not need to borrow to make early health investments; in addition, the redistributive effect of the policy allows them to borrow less for consumption purposes. Second, due to the complementarity of investments, the increase in early health level increases the productivity of later educational investments. This pushes poor youths to borrow additional funds to invest in their education—particularly those with high learning ability. Among developing countries, the first effect dominates the second, resulting in nearly a 10% reduction in the fraction of families that are financially constrained. However, in the developed world the second effect dominates resulting in over a 7% *increase* in constrained families. These results are consistent with the previous discussion on features of the baseline economies. Specifically, families in higher income countries primarily borrow funds to finance education while those in developing countries also borrow for consumption and early health investments.

My second main finding is that early health gaps are an important source of schooling variation *across* countries. Raising to equate early health within countries results in a 19.2% reduction in the cross-country standard deviation of schooling (see Table 1.8). Schooling inequality *within* countries also looks more similar *across* countries—the cross-country standard deviation of schooling Ginis falls by 36.8%. This further highlights that early health inequalities have differing effects on schooling gaps across national income levels. However, equating early health within countries with a tax and transfer scheme has little effect on the world log-income distribution. In my next experiment, I compare how these results differ when exogenously equating early health worldwide to the average U.S. level.

Table 1.8: Equating Health *Within* Countries

| Cross-Country S.D. % Change | | |
|-----------------------------|--------------|-----------------|
| <i>Ave sch</i> | <i>log y</i> | <i>sch Gini</i> |
| -19.2 | 0.0 | -36.8 |

1.6.2 Equating Early Health *Across* Countries

The primary purpose of this experiment is to quantitatively assess how *cross-country* differences in early health levels affect the world dispersion of schooling attainment. To this end, I equate early health investments i to the average baseline level of the U.S. for all individuals in all countries. As opposed to the previous experiment, I leave tax rates at their baseline level for all countries, as much of the developing world would not be able to internally fund such a large change in early health investments. The main results of the experiment are reported in Table 1.9 and illustrated in Figure 1.13.

Table 1.9: Equating Health *Across* Countries

| Cross-Country S.D. % Change | | |
|-----------------------------|--------------|-----------------|
| <i>Ave sch</i> | <i>log y</i> | <i>sch Gini</i> |
| -53.4 | -12.7 | -40.2 |

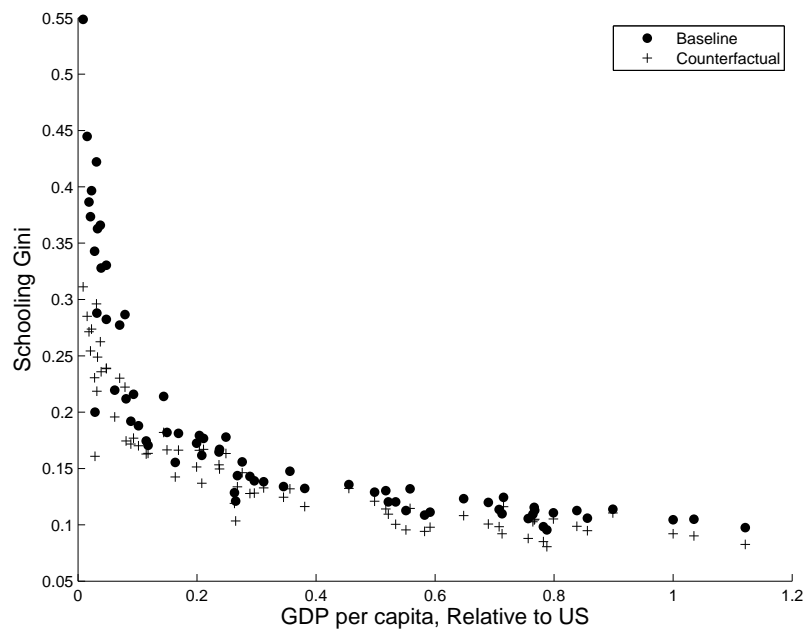
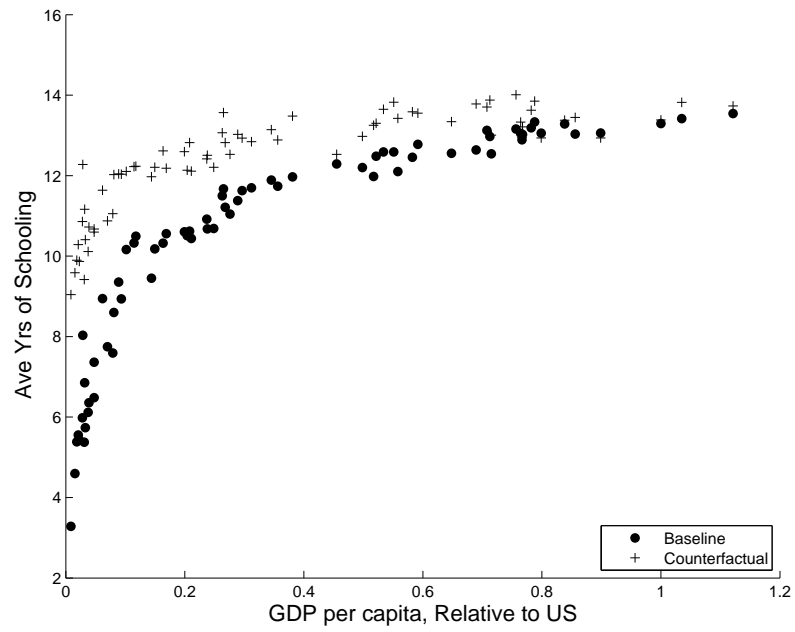


Figure 1.13: Equating to Ave. U.S. Early Health

As a result of the experiment, the cross-country standard deviation of schooling falls by 53.4%, suggesting that early health differences *across* countries are an important source of observed schooling attainment gaps. Early health inequalities also play a relatively significant role in determining the world income distribution—the standard deviation of log-output falls nearly 13%. The cross-country standard deviation of schooling Ginis falls by 40%, which is only slightly larger than the previous experiment equating early health levels within but not across countries. This occurs because schooling Ginis fall considerably more for the highest income countries relative to the first experiment. Equating early health to the *average* U.S. level for everyone results in lower levels of early health for many wealthy, high-ability youths in high income countries. Due to complementarities, these youths choose to reduce schooling nearly as much as poor or lower-ability youths increase schooling. This decreases schooling Ginis in the richest countries but has little effect on average attainment levels.

The average within country effects of the experiment are shown in Table 1.10. Average schooling levels in developing countries increase by nearly 50% while the average schooling Gini falls by 18.9%. Notice that once again output rises and consumption inequality within countries falls as a result of equating health among children. However, since this experiment no longer requires countries to fund the increase in early health investments with higher taxes, the increase in output is accompanied by a large increase in consumption, especially among developing countries. Finally, note that developing countries again experience an average decrease in the fraction of families constrained due to the level and redistributive effects of the experiment.

Table 1.10: Equating Health *Across* Countries (Ave % Change)

| Countries | <i>School</i> | | <i>log y</i> | <i>Consumption</i> | | % <i>Constrained</i> |
|------------|---------------|-------------|--------------|--------------------|-------------|----------------------|
| | <i>Ave</i> | <i>Gini</i> | | <i>Ave</i> | <i>Gini</i> | |
| All | 26.1 | -14.4 | 2.9 | 28.7 | -12.3 | -4.6 |
| Developing | 48.2 | -18.9 | 5.0 | 48.0 | -15.7 | -12.0 |

1.6.3 Education and Early Health Subsidies

I next discuss two alternate policy experiments to compare the effects of an increase in early health subsidies versus a roughly cost-equivalent increase in later education subsidies. Specifically, I increase the annual tax rate the equivalent of \$30 per youth (\$90 per child) based on baseline output levels and increase either early health or education subsidies accordingly. For example, if a country's baseline annual output was valued at \$3,000 per youth, I increase the tax rate by 1% and devote these additional public funds to either early health or education subsidization.

Recall that due to data availability issues on food subsidies, explicit public health spending in the baseline was restricted to only medical care subsidies p_m . However, for the early health subsidy experiment, I keep medical care subsidies fixed at baseline levels and assume that the additional public funds are used to subsidize composite early health investments i at the rate p_i . This is equivalent to assuming that the government divides the new funds optimally between food and medical care subsidies. I denote the tax rate designated for the composite health subsidy τ_i . Note that it is the tax rates (τ_i, τ_q) that are exogenously adjusted in these experiments, while the actual subsidy rates (p_i, p_q) are determined endogenously by ensuring that the government budget is balanced in the new stationary equilibrium. As detailed below, the tax increase of \$30 per youth has a substantial impact among developing countries. However, this dollar amount represents a very minimal increase in public funding among richer countries and effects are very mild. As such, I restrict my reporting and discussion of the policy effects to developing countries. Results of both experiments for these countries are summarized in Table 1.11.

Table 1.11: Increasing Subsidies in Developing Countries (Ave % Change)

| Experiment | i | | s | | q | | $\log y$ | c | | % |
|-------------------|------------|-------------|------------|-------------|------------|-------------|----------|-------------|--------------------|------|
| | <i>Ave</i> | <i>Gini</i> | <i>Ave</i> | <i>Gini</i> | <i>Ave</i> | <i>Gini</i> | | <i>Gini</i> | <i>Constrained</i> | |
| Increase τ_i | 53.1 | -5.5 | 16.0 | -9.3 | 20.2 | -3.9 | 1.4 | -4.6 | | -7.4 |
| Increase τ_q | 3.5 | 0.2 | 6.4 | -10.4 | 32.0 | -5.6 | 0.4 | -0.5 | | 0.2 |

Devoting the additional public funds towards early childhood health raises average investments i by 53% and reduces the corresponding Gini by 5.5%. Due to dynamic complementarity, increasing early investments also results in substantial increases of human capital investments in *later* stages of child development. Specifically, there is a 16% increase in average years of schooling and a 20% increase in average educational quality investments. Furthermore, the inequality of investments is reduced along each of these dimensions. All together the policy change results in a 1.4% average increase in log-output, a 4.6% average decrease in consumption inequality, and an average of 7.4% fewer constrained families.

When the increased public funding goes directly to education, there is a substantial 32% average increase in educational quality investments accompanied by a 6.4% average increase in schooling. Inequality along these two dimensions falls a similar magnitude as the previous experiment. However, early health investments increase a much lower 3.5% and the early health Gini *rises* a very small 0.2% on average. Intuitively, it is the wealthier unconstrained parents who are able to increase their early health investments to fully take advantage of the anticipated benefit of the more generous education subsidy when their child becomes a youth the next period. Conversely, while poor constrained families invest more in educational quality because it is cheaper, they are unable to adjust earlier investments in health to fully take advantage of complementarities. As a result, output rises and consumption inequality falls to a much lesser extent than when new public funds are spent on early childhood health. This implies there is no equity-efficiency trade-off between the two alternate policy options. Increasing subsidies to early health results in more output and less inequality than a cost-equivalent increase in educational subsidies.

The main policy conclusion drawn from these experiments is that the effects of early health interventions tend to be amplified by later educational choices. At the same time, interventions that target school-aged children may be limited if early health conditions are not taken into account. This is not to conclude that the reduction of public education funding in favor of health spending is a mechanism to improve child outcomes. Rather, an

alternate policy perspective is that the effects of public education in the developing world could be most effectively amplified if combined with additional early health interventions.

1.7. CONCLUSIONS

In this chapter I developed a theory of human capital accumulation to quantitatively assess the importance of early childhood health in determining later educational investments. After restricting the parameters governing the production of human capital with data from the U.S., I use the theory to examine what drives the observed variation of schooling attainment across countries. The calibrated model finds strong complementarity between early childhood health investments and later educational investments. As a result, early childhood health inequalities are amplified into large schooling differences both *within* and *across* countries.

Quantitatively, I find that raising early health investments to equalize levels *within* countries lowers schooling Gini coefficients an average of 12% in developing economies but has very mild effects in richer countries. The experiment also results in over a 19% reduction in the cross-country standard deviation of average schooling attainment. Furthermore, when early health is equalized worldwide to the average U.S. level, the cross-country dispersion of schooling falls over 53% relative to the baseline. This quantitatively demonstrates the importance of early health differences in explaining the large and persistent schooling gaps observed across countries.

Due to the dynamic complementarity of investments, I also find that the timing of policy intervention has important implications for human capital accumulation over childhood. In counterfactual experiments I find that increasing subsidies to early childhood health has substantially larger effects on investment decisions than later subsidies to education. These findings reinforce the notion of child health policy as an effective educational policy tool.

Finally, my model is able to reproduce the cross-country dispersion of schooling attain-

ment and inequality quite well by incorporating cross-country heterogeneity in public health and education subsidies, prices, and financial development. Public expenditures help alleviate underinvestment by the poor while financial development governs the extent to which parents can borrow against future earnings to invest in themselves or their children. Clearly other factors affect schooling differences across countries, but I abstract from these in my current research to focus on the above channels. In future work, I plan to extend my analysis along multiple dimensions to explore how cross-country differences in fertility, life expectancy, and child mortality rates interact with dynamic health and educational investment decisions.

2. THE NONCOGNITIVE RETURNS TO PRIVATE SCHOOLING IN INDIA: EVIDENCE ON SELF-EFFICACY AND SELF-ESTEEM¹

2.1. INTRODUCTION

Research from a variety of disciplines has shown that school context and the development of noncognitive traits (or psychosocial competencies) are theoretically and empirically related. While evidence suggests that school environment directly influences noncognitive development, the magnitude and underlying causal mechanisms of the relationship remain an active area of research. Predominately, existing work has been focused on children transitioning through adolescence in economically advanced countries (e.g. Usher and Pajares 2008; Morin et al. 2013). However, recent economic research finds that educational investments made during earlier stages of the life-cycle may be particularly influential in the development process (Cunha and Heckman 2007; Cunha et al. 2010). Moreover, substantial diversities in school environments have been documented in developing countries such as India (e.g. Galab et al. 2013; Muralidharan and Sundararaman 2015). As such, this chapter contributes to the

¹The data used in this chapter come from Young Lives, a 15-year study of the changing nature of childhood poverty in Ethiopia, India (Andhra Pradesh), Peru and Vietnam (www.younglives.org.uk). Young Lives is core-funded by UK aid from the Department for International Development (DFID) and co-funded from 2010 to 2014 by the Netherlands Ministry of Foreign Affairs. The views expressed here are those of the author. They are not necessarily those of Young Lives, the University of Oxford, DFID or other funders.

literature by examining the effect of school context on the early development of psychosocial competencies in a less advanced economy. Specifically, I explore the impact of attending private school in the Indian state of Andhra Pradesh on age eight measures of self-efficacy and self-esteem. By exploring the effect of private schools on early psychosocial competencies, this chapter begins to uncover which features of educational context may (or may not) be most important for the development of noncognitive traits in India and in the broader developing world.

Private school enrollment in India has substantially grown over the past several decades—estimated enrollments are now over 28% in rural areas and 65% in urban (Pratham 2013; Rangaraju et al. 2012). With the passing of the Right to Education Act in 2009, this trend is likely to continue.² At the same time, there is growing evidence that there are positive but relatively modest cognitive returns to private schooling in India (French and Kingdon 2010; Singh 2013; Muralidharan and Sundararaman 2015). The revealed preference for private schooling combined with the evidence on cognitive returns suggests that there is scope for possible gains along noncognitive dimensions. Moreover, Indian data from the Young Lives Study (YLS) reveals that eight year olds enrolled in private school score significantly higher than their public school counterparts on measures of self-efficacy and self-esteem—two related but distinct facets of individual personality (see Section 2.4.1). However, these gaps show only an overall correlation between private school and noncognitive traits. My aim in this chapter is to explore the causal effects of private school on these psychosocial competencies, and how they may vary across different populations of children.

The effects of interest are estimated using a semiparametric structural model of private school choice where selection occurs based on observable child and family characteristics as well as unobserved factors. Specifically, I model unobserved factors as child endowments of cognitive skill, health, and social skill. While well accepted in epidemiology, only recently have economists begun to explore the notion that life outcomes are influenced by the interplay

²The law makes primary education a fundamental right and requires private schools to reserve 25% of capacity for enrollment of disadvantaged populations.

of cognitive, physical, and social and emotional traits—a concept referred to as developmental health (Conti et al. 2011). I follow the work of Carneiro et al. (2003) and Hansen et al. (2004) by using Bayesian Markov Chain Monte Carlo (MCMC) methods to simulate the parameters of the model.

I estimate the average treatment effect of attending private school between age five and eight as an increase in self-efficacy and self-esteem measures of 0.15 and 0.28 standard deviations, respectively. These results are within the range of credible estimates of effects on cognitive outcomes in India, suggesting that private school may play at least as significant a role in early psychosocial as in cognitive development. My estimation strategy also allows for the identification of the distribution of treatment effects across family and child characteristics. Understanding which children may or may not benefit from attending private school is important to help policymakers target interventions in the most efficient manner and may also shed further light on the underlying mechanisms at work. I find that effects differ by observable characteristics such as gender, urbanization, caste, household wealth, and maternal measures of self-efficacy and self-esteem. I also find that effects may differ between children who are similar in their observed characteristics based on unobserved differences in cognitive skill, health, and social skill.

In addition to contributing to the literature relating school environment to psychosocial development, this chapter also adds to the research on the returns to private schooling in developing economies. Although to my knowledge no other research examines the effects of private school on noncognitive traits, studies in developing countries have found mixed evidence on the effect of private schooling on cognitive outcomes (e.g. Angrist et al. 2002; Hsieh and Urquiola 2006; Newhouse and Beegle 2006; Bold et al. 2013). Within India, several have found moderate positive effects (French and Kingdon 2010; Singh 2013). In the most rigorous Indian study, Muralidharan and Sundararaman (2015) evaluate the effects of a randomized offer of private school vouchers. Coincidentally, the experiment takes place in the same state and for the same age cohort as the YLS data used in this chapter. The

authors find no effect of private school on math and Telugu (the native language) but positive effects on English, Science, Social Studies, and Hindi. The estimated combined effect across test subjects is similar in magnitude to those found on self-efficacy and self-esteem in this chapter.

The remainder of this chapter is presented as follows: in Section 2.2, I discuss related literature on psychosocial development and the state of private schooling in India. In Section 2.3, I develop the structural model of school choice and discuss the identification and estimation strategy. Section 2.4 discusses the data, while Section 2.5 describes simulation algorithms and empirical results. In Section 2.6, I present the results of a counterfactual analysis related to the Rights to Education Act of 2009. Finally, Section 2.7 concludes.

2.2. SELF-EFFICACY, SELF-ESTEEM, AND PRIVATE SCHOOLING IN INDIA

In recent years, economists have paid increasing attention to the importance of noncognitive skills on a variety of economic and behavioral outcomes.³ In this chapter, I focus on the effects of private school attendance in India on composite measures of self-efficacy and self-esteem—two related but distinct facets of individual personality that have been extensively studied in the field of psychology. Self-efficacy relates closely to the psychological concept of locus of control, or sense of agency or mastery over one’s own life. Individuals with a high level of self-efficacy maintain a strong belief that outcomes are a result of their own efforts, as opposed the action of others, fate, or random luck. Many studies have found self-efficacy (or an internal locus of control) to have positive effects on behavioral outcomes such as educational attainment or subjective belief of the probability of finding a job (e.g. Coleman

³For an overview of related literature and the interface between personality psychology and economics, see Borghans et al. (2008).

and DeLeire 2003; Caliendo et al. 2010). The results on wages and other labor outcomes are more mixed but still find generally positive effects (e.g. Andrisani 1981; Heineck and Anger 2010; Piatek and Pinger 2010). In contrast to self-efficacy, self-esteem reflects an individual's overall sense of self-worth. In the psychology literature, self-esteem has been shown to have strong correlations with a variety of outcomes both in and out of the labor market (e.g. wages, health, life satisfaction, education). While evidence of causality is heavily debated for many labor market and health outcomes, there is stronger support that self-esteem leads to greater happiness (Baumeister et al. 2003; Trzesniewski et al. 2006).

There is increasing evidence that schooling is related to the development of noncognitive traits in general. For example, researchers have found that schooling attainment affects adult outcomes believed to be heavily influenced by psychosocial competencies such as health and health behaviors, criminal activity, teen fertility, political engagement, and life satisfaction (e.g. Emler and Frazer 1999; Conti et al. 2011; Oreopoulos and Salvanes 2011). Recent theoretic and empirical work by James Heckman and coauthors suggests that human capital investments such as schooling contribute to the evolution of both cognitive and noncognitive skills by building off of existing abilities and personality (Heckman 2006; Cunha and Heckman 2007; Cunha et al. 2010). These papers also find evidence that child development is more sensitive in the early stages of the life-cycle and document the substantial impact of many early childhood interventions on behavioral outcomes.

While economists have established an important relationship between schooling and noncognitive skill development, the psychology literature provides several theories of the underlying mechanisms at work. A prominent example is the work of Albert Bandura (1986; 1997), who hypothesized that children interpret information from four sources which contribute to the evolution of self-efficacy: mastery experience (e.g. successful completion of a task), vicarious experience (e.g. social models or comparison to others), verbal and social persuasion (e.g. parent or teacher encouragement), and emotional and physiological states (e.g. anxiety, fatigue, mood). While mastery experience is considered the most influential

source of self-efficacy, vicarious experience is believed to play a more prominent role when transitioning into adolescence (e.g. from elementary to middle school) as children become more attune to peer group comparisons. In contrast, it is believed that verbal and social persuasion plays a more prominent role during the earlier formative years when children are more attentive to messages received from mentor figures such as parents and teachers. Research also suggests that it may be easier to undermine a child's self-efficacy through social persuasion than to increase it (e.g. through negative teacher feedback). A wealth of related research shows that school context may play a role in the early development of self-efficacy, for example, through facilitating the successful completion of tasks as well as verbal and social persuasion of teachers or other staff.⁴

The development of self-esteem has also been shown to have a strong relationship with school context. While few studies have focused exclusively on the early development of self-esteem, many have examined the effect of school environment on adolescent self-esteem. For example, studies have found school climates that promote fairness, security, student autonomy and participation, and positive interpersonal relationships with teachers and peers have a positive association with self-esteem (Hoge et al. 1990; Hirsch and DuBois 1991; Deihl et al. 1997; Roeser and Eccles 1998; Reddy et al. 2003; Greene and Way 2005; Way et al. 2007; Morin et al. 2013). Student's academic achievement as well as perception that the school climate promotes mastery and achievement are also related to self-esteem (Hoge et al. 1990; Hirsch and DuBois 1991; Zimmerman et al. 1997; Roeser and Eccles 1998; Morin et al. 2013).

Drawing on the psychology literature on psychosocial development, private schools in India could conceivably influence self-efficacy and/or self-esteem through an increase in academic achievement. As several studies have found positive (albeit moderate) effects of private school on a number cognitive outcomes, this seems at least one plausible channel of influence (French and Kingdon 2010; Singh 2013; Muralidharan and Sundararaman 2015). However,

⁴For a comprehensive review of the evidence linking school context to self-efficacy, see Usher and Pajares (2008)

private schools could be influencing psychosocial competencies through other channels as well. Private and government-run schools in India are different along many observed (and likely unobserved) dimensions that could conceivably influence noncognitive development. For example, quantitative data show that private schools experience lower teacher absence, better infrastructure, better school hygiene, longer school days, and longer school years (Galab et al. 2013; Muralidharan and Sundararaman 2015).

Qualitative YLS data on parent perceptions of school environment further supports the possibility that private school directly influences noncognitive development.⁵ For example, one parent sending children to private school claimed to have “observed a change in their attitudes, they are now more responsible towards their homework and they are using cultured language.” Other parents perceived private schools to have a higher overall quality of instruction, including teachers giving more individual attention and care:

In [a] government school, the teaching imparted is below standards... they don't pay individual attention and classes are so irregular and lessons are not taught properly... whereas in private schools teaching is good and teachers are taking individual care [for] each and every student.

Perceived lack of quality in government-run schools is also driven by higher rates of teacher absence: “in government schools, teaching one day... take 10 days break... they don't take any care at all... but in private schools we pay and they take much care”. Other parents noted teacher punctuality and efforts to connect with parents as major drivers of private school enrollment:

I observed that teachers in public schools are coming [to class] very late... they don't bother even if [a] child [is] absent for [a] number of days... in private school, where my two children are studying, teachers are on time to class and if any child [is] absent for a day they will inquire with parents... such concern is important for us.

⁵Source of translated qualitative data: Kumar 2013.

All of these actual or perceived deficiencies in instructional quality in government-run schools could weaken interpersonal relationships and/or result in ineffective (or negative) verbal and social persuasion, ultimately stunting or regressing psychosocial development.

A final important contextual distinction is that private schools are more likely to use English as a medium of instruction than their government-run counterparts, particularly during early grades. As one parent noted, “we must give English medium education to our children because it only gives better job opportunities.” Parent responses also suggest that the availability of English medium during the early stages of private schooling may be particularly valued. For example, one parent stated that if a child is first exposed to “Telugu medium and if we change them later to English medium it will not work out and they [will] suffer. . . so from the beginning we want them to have a strong [English] foundation.” If using English as a medium of instruction is perceived to increase future academic or career success, this may be another important channel through which private schools influence psychosocial competencies.

2.3. MODEL

Consider a simple environment where the development of a child’s psychosocial competencies is dependent on a parent’s choice to send them to private school or not. Specifically, in my model, psychosocial outcomes for each child differ based on the type of school chosen. Following the factor structure of Carneiro et al. (2003) and others, school choice and subsequent outcomes are assumed to be functions of observed family and child characteristics as well as a vector of latent factors (θ). These factors are known to the parent but are imperfectly observed by the researcher. This allows parents to sort children across schooling type based on observed and unobserved characteristics. I model and interpret these unobserved factors as child endowments of cognitive skill, health, and social skill: $\theta = (\theta_C, \theta_H, \theta_S)$. Identifica-

tion and interpretation of the unobserved factors relies on the inclusion of a set of imperfect measurements that are not affected by the relevant schooling choice made by the parent. It is also important to note that included in the observed family characteristics is a set of maternal psychosocial competencies. These measures serve to control and allow for examination of the intergenerational transmission of psychosocial traits.

2.3.1 Schooling Choice

The choice of sending a child to private school or not is modeled using a standard latent index structure. Let the observed binary choice be given by $P_i = 1$ if child i attends private school and $P_i = 0$ otherwise. Furthermore, let P_i^* be the net utility from choosing private school and characterize the decision rule:

$$P_i = \begin{cases} 1 & \text{if } P_i^* > 0 \\ 0 & \text{otherwise} \end{cases}.$$

For empirical analysis, net utility is modeled as a linear function of observed and unobserved characteristics:

$$P_i^* = \gamma Z_i + \lambda_P \theta_i + \epsilon_{Pi},$$

where Z is a vector of observed covariates, θ is a vector of unobserved factors, and ϵ_P is an idiosyncratic error term. Observed characteristics, unobserved factors, and the error term are assumed to be statistically independent of each other: $Z \perp \theta \perp \epsilon_P$.

2.3.2 Outcomes

For each child, it is assumed there are two possible realizations for each outcome of interest. A potential outcome if the child attends private school (Y_i^1) and a potential outcome if they attend a government-run school (Y_i^0). In the empirical implementation of the model, the observed outcomes are discrete and ordinal in nature. As such, I again assume a latent index

structure with each underlying potential outcome (Y_i^*) linearly dependent on observed and unobserved characteristics according to:

$$Y_i^{*t} = \beta^t X_i + \lambda_Y^t \theta_i + \epsilon_{Y_i}^t \quad \text{for } t = 0, 1$$

where X is a vector of observed covariates and $X \perp\!\!\!\perp \theta \perp\!\!\!\perp \epsilon_Y^1 \perp\!\!\!\perp \epsilon_Y^0 \perp\!\!\!\perp \epsilon_P$. The index is discretized into V ordered values by a set of cut-points (κ) to produce the potentially observed discrete outcomes:

$$Y_i^t = v \quad \text{if } \kappa_{t,v-1} < Y_i^{*t} < \kappa_{t,v} \quad \text{for } t = 0, 1, \quad v = 1, \dots, V.$$

While a child can potentially realize either outcome ($t = 0, 1$), the observed outcome depends on the choice of schooling type. Specifically, using switching regression notation (Quandt 1972), the observed outcome for any child can be written:

$$Y_i = P_i Y_i^1 + (1 - P_i) Y_i^0.$$

2.3.3 Endowment Measurement System

In order to identify the joint distribution of the unobserved factors θ , the model includes a system of imperfect endowment measurements that are observed prior to treatment. Following Conti et al. (2011), each measurement is assumed to be a function of observables and a dedicated unobserved endowment. Dedicated measures imply that, for example, height only measures a child's health and not their endowment of cognitive or social skill. This does not imply, however, that an individual's endowments are unrelated, as they are allowed to be correlated— $Cov(\theta_{i,j}, \theta_{i,k}) \neq 0, \forall j \neq k$.⁶ This measurement system not only allows for identification, but also helps give interpretation to the unobserved factors by anchoring them

⁶Identification of a correlated factor model using the measurement system described in the empirical analysis can be shown with slight modification to the proof provided in Conti et al. (2011).

in observed data.

In the empirical implementation of the model, measures of cognitive skill (M_C) and health (M_H) are continuous and assumed to follow a linear structure:

$$M_{Cn,i} = \delta_{Cn}W_i + \lambda_{Cn}\theta_{C,i} + \epsilon_{Cn,i} \quad \text{for } n = 1, \dots, N_C$$

$$M_{Hn,i} = \delta_{Hn}W_i + \lambda_{Hn}\theta_{H,i} + \epsilon_{Hn,i} \quad \text{for } n = 1, \dots, N_H$$

where W is a vector of observed covariates, N_C the number of cognitive measures, and N_H the number of health measures. The social skill measures (M_S) are ordinal or binary; thus I assume a latent index structure given by:

$$M_{Sn,i}^* = \delta_{Sn}W_i + \lambda_{Sn}\theta_{S,i} + \epsilon_{Sn,i} \quad \text{for } n = 1, \dots, N_S$$

such that the observed value is given by:

$$M_{Sn,i} = v \quad \text{if } \kappa_{v-1} < M_{Sn,i}^* < \kappa_v \quad \text{for } v = 1, \dots, V_n$$

where V_n is the number of possible ordinal values for social skill measure M_{Sn} .

The scale of each of factor is set by normalizing the factor loading in the first measurement of each endowment to one ($\lambda_{C1} = 1, \lambda_{H1} = 1, \lambda_{S1} = 1$). Finally, identification requires that the idiosyncratic errors in the measurement system are statistically independent from each other and from the errors in the school choice and outcome equations: $\epsilon_{Cn} \perp\!\!\!\perp \epsilon_{Hn} \perp\!\!\!\perp \epsilon_{Sn} \perp\!\!\!\perp \epsilon_Y^1 \perp\!\!\!\perp \epsilon_Y^0 \perp\!\!\!\perp \epsilon_P, \forall n$.

2.3.4 Identification and Estimation

Identification of the model is based on the following conditional independence assumption:

$$(Y^0, Y^1) \perp P \mid Z, X, W, \theta.$$

Notice that this is the same assumption used in standard matching strategies. The difference between standard matching and the strategy used in this chapter lies in the fact that factor endowments are imperfectly observed and are instead approximated with error by a system of observed measurements.

A number of additional assumptions are made in order to estimate the model empirically. First, I assume the latent factors follow a multivariate normal distribution given by:

$$\theta \sim N(\mu, \Sigma)$$

where μ is a 3×1 vector and Σ is a 3×3 matrix.⁷ The variance-covariance matrix is not restricted to be diagonal as a child's latent factors are allowed to be correlated. Second, idiosyncratic errors on all outcome equations, the choice equation, and discrete measurements are assumed to be distributed normally with mean zero and variance one. This is the usual solution for keeping latent response variables invariant to scale transformations. For continuous measurements, errors are assumed to be distributed normally with mean zero and unknown variance.

The density of outcomes over unobservables can be written:

$$\int \int \int_{\theta_C, \theta_H, \theta_S} f(Y, P, M_C, M_H, M_S \mid Z, X, W, t_C, t_H, t_S) dF_{\theta}(t_C, t_H, t_S)$$

where $F_{\theta}(\cdot)$ denotes the joint cumulative distribution of latent factors and $f(\cdot)$ denotes the joint density of schooling choices, outcomes, and measurements. Note that conditional on observables and unobservables, private schooling choice P and all measurements M are independent.

Bayesian Markov Chain Monte Carlo (MCMC) methods are used to estimate the model parameters for computational convenience. I follow the procedures discussed in Carneiro et al. (2003) and Hansen et al. (2004) for estimation.⁸ The key feature of the procedure

⁷A mixture of multivariate normals was also used to more flexibly approximate the distribution of latent factors, but the improvement in the fit of the model was negligible.

⁸I run 50,000 iterations of the Gibbs sampler and, after discarding the first 10,000 as a burn-in period,

is that latent factors are drawn from their joint posterior distribution in each iteration for each individual. The factor draws are subsequently used to update the other model parameters including those of the joint distribution of factors itself. Not only is the procedure computationally attractive, it also provides a distribution of parameter estimates which are useful in simulating counterfactual choices and outcomes.

2.4. DATA

I use data from the Young Lives Study (YLS) which conducted surveys for a cohort of 2,011 children born January 2001 to June 2002 in the Indian state of Andhra Pradesh. Currently data are available from three rounds of surveys conducted in 2002, 2006-07, and 2009-10, when children were approximately one, five, and eight years old, respectively. The study collected detailed information on household and child characteristics, including questions on both parent and child perceptions, attitudes, and feelings in addition to multiple rounds of child cognitive test scores and health measures. After removing observations with missing outcomes or covariates, I am left with a sample of 1,856 observations. Throughout the empirical analysis, classification into the private school “treatment” group is defined as having attended a private school in the 2007-08 and/or 2008-09 academic years.⁹

2.4.1 Psychosocial Outcomes

The outcomes of interest are based on a child’s agreement to a number of statements related to self-efficacy and self-esteem during the 2009-10 collection cycle, when children were approximately eight years old. Agreement with each statement is measured on a Likert scale ranging from 1 (“strongly disagree”) to 5 (“strongly agree”). The statements are based on

include one out of every 40 iterations in the posterior estimates.

⁹Results are robust to alternate specifications of the treatment group.

the educational psychology literature but were adapted, extensively pilot tested, and subjected to psychometric validation by the YLS. The self-efficacy measure is based on the statements: “If I try hard, I can improve my situation in life,” “I like to make plans for my future studies and work,” and “If I study hard at school, I will be rewarded by a better job in the future.” The statements relating to self-esteem are centered around the concepts of pride and shame: “I am proud of my shoes or of having shoes,” “I am proud of my clothes,” and “I am proud that I have the correct uniform.” Assuming the statements measure a single underlying factor is broadly validated in Dercon and Krishnan (2009), and I obtain similar Cronbach’s alphas of 0.57 and 0.69 for the self-efficacy and self-esteem statements, respectively.¹⁰ As such, a single score is created for each outcome by averaging non-missing responses across the three corresponding statements. However, due the limited number of statements, the raw outcome measures take on a relatively small number of discrete values. Furthermore, there is clear truncation on either end of the measures, suggesting that treating them as continuous could result in significant bias. As such, I treat observed outcomes as ordinal representations of an underlying latent index. In order to the reduce the computational burden of estimation, ordinal values with less than 50 observations are combined with the closest neighboring value. This results in a total of seven and nine ordinal values for the self-efficacy and self-esteem measures, respectively. Table 2.1 shows that eight year olds enrolled in private school score significantly higher than their public school counterparts on these raw measures of self-efficacy and self-esteem (see also Figure 2.1 for the distribution across values).

2.4.2 Observed Covariates

A number of observed covariates are included in the outcome, measurement, and choice equations to control for family background and other child characteristics. In all equations,

¹⁰It is commonly suggested a Cronbach’s alpha close to at least 0.7 is required for a “reliable” measure (i.e. the items measure a single underlying factor). However, a small number of items and measurement error also serve to lower Cronbach’s alpha.

Table 2.1: Psychosocial Outcomes at 8 Years of Age by School Type: Raw Scores

| | Private School | | | Public School | | | <i>z</i> -statistic | <i>p</i> -value |
|---------------|----------------|-------|-----|---------------|-------|-------|---------------------|-----------------|
| | Mean | S.D. | n | Mean | S.D. | n | | |
| Self-efficacy | 4.25 | 0.529 | 773 | 4.00 | 0.569 | 1,083 | -9.12 | 0.000 |
| Self-esteem | 4.07 | 0.741 | 773 | 3.60 | 0.795 | 1,083 | -13.02 | 0.000 |

Source: Young Lives Study, India, younger cohort. Statements measured on Likert scale ranging from 1 to 5. Individual's raw score calculated by averaging non-missing responses across coresponding statements. Statistics reported from a two-sample Wilcoxon rank-sum (Mann-Whitney) test.

I include a wealth index, number of siblings, father's education, mother's education, caste, urban status, a set of region dummies, as well as the child's gender and age in months. I also include four psychosocial competency indices for the mother or primary caregiver of the child: self-efficacy, self-esteem, inclusion, and trust.¹¹ Similar to the child outcomes detailed above, these indices are derived from agreement to a series of statements relating to each psychosocial competency. However, unlike the child outcomes, there are enough statements and variation in responses across statements to construct approximately continuous measures. Specifically, the responses to each statement are standardized to create a set of *z*-scores with mean zero and variance one. The composite indices are then created by taking the average score across non-missing *z*-scores for each individual. The availability of such noncognitive measures for both parent and child is one of the unique strengths of the YLS data. These indices not only control for possible private school selection based on parental competencies, but also allow for examination of the magnitude and channels through which psychosocial traits may be transmitted from one generation to the next.

Lastly, I assume that in addition to the covariates above and unobserved child endowments, school choice is also affected by the relative distance to a private versus a public school. Specifically, in the choice equation I include a measure of how much longer it takes (in minutes) to travel to a private versus a public school from the center of a child's home community.¹² Summary statistics for all observed covariates are reported in Table 2.2.

¹¹Mothers were the primary caregiver for over 95% of the sample.

¹²This measure is truncated at ± 60 minutes.

Table 2.2: Summary Statistics: Observed Covariates

| | Total | | Private School | | Public School | |
|------------------------------|-------|------|----------------|------|---------------|------|
| | Mean | S.D. | Mean | S.D. | Mean | S.D. |
| Male | 0.53 | 0.50 | 0.60 | 0.49 | 0.48 | 0.50 |
| Wealth Index | 0.45 | 0.19 | 0.58 | 0.18 | 0.37 | 0.15 |
| Age | 5.36 | 0.32 | 5.36 | 0.33 | 5.36 | 0.32 |
| Siblings | 1.55 | 1.07 | 1.31 | 0.95 | 1.72 | 1.12 |
| Father Years Education | 5.68 | 5.15 | 8.37 | 5.09 | 3.71 | 4.21 |
| Mother Years Education | 3.72 | 4.51 | 6.10 | 4.96 | 1.97 | 3.17 |
| Urban | 0.24 | 0.43 | 0.48 | 0.50 | 0.07 | 0.26 |
| Scheduled Caste | 0.18 | 0.39 | 0.13 | 0.33 | 0.23 | 0.42 |
| Scheduled Tribe | 0.13 | 0.34 | 0.06 | 0.23 | 0.18 | 0.38 |
| Other Backward Classes | 0.48 | 0.50 | 0.48 | 0.50 | 0.49 | 0.50 |
| Mother Self-efficacy | 0.00 | 1.00 | 0.26 | 0.97 | -0.18 | 0.99 |
| Mother Self-esteem | 0.00 | 1.00 | 0.28 | 0.97 | -0.21 | 0.96 |
| Mother Trust | 0.00 | 1.00 | -0.02 | 1.00 | 0.01 | 0.99 |
| Mother Inclusion | 0.00 | 1.00 | 0.21 | 1.01 | -0.17 | 0.96 |
| Add'l Min. to Private School | 16.5 | 23.1 | 8.9 | 19.6 | 21.9 | 23.9 |

Source: Young Lives Study, India, younger cohort. Includes all observations without missing covariates or outcomes (n=1,856).

2.4.3 Endowment Measurements

As measures of cognitive endowment, I use two tests administered during the second round of data collection (age five)—the Peabody Picture Vocabulary Test (PPVT) and the Cognitive Development Assessment (CDA) quantitative subscale. The PPVT is a widely used test of receptive vocabulary, while the CDA was designed by the International Evaluation Association to assess cognitive development in four year olds. Validation of both measures as used in the YLS are discussed in Cueto et al. (2009). As dedicated measures of latent child health, I use height-for-age z-scores collected in the second round as well as weight-for-age z-scores from both round one and two (approximately age one and five, respectively).¹³

During the second round of data collection, the parent or primary caregiver of each child

¹³Health measure z-scores are derived by the YLS by standardizing against an international reference population.

was asked about all activities carried out by the child during the most recent or typical workday. Social endowment measures are derived from these responses and center around a child’s interaction with other children. Specifically, as the first social endowment measure I use the total number of activities the child completed with siblings, friends, or other children (e.g. chores, playing, visiting relatives, watching TV). The second measure is an binary indicator of whether or not the child spent time playing with friends explicitly (excludes play with siblings or other children). Summary statistics for all measurements are reported in Table 2.3.

Table 2.3: Summary Statistics: Endowment Measurements (Age 5)

| | Total | | | Private School | | | Public School | | |
|---------------------------|-------|------|----------|----------------|------|----------|---------------|------|----------|
| | Mean | S.D. | <i>n</i> | Mean | S.D. | <i>n</i> | Mean | S.D. | <i>n</i> |
| <i>Cognitive Measures</i> | | | | | | | | | |
| PPVT (vocabulary) | 0.00 | 1.00 | 1,772 | 0.26 | 1.13 | 736 | -0.18 | 0.85 | 1,036 |
| CDA (cognitive) | 0.00 | 1.00 | 1,845 | 0.27 | 0.93 | 771 | -0.19 | 1.00 | 1,074 |
| <i>Health Measures</i> | | | | | | | | | |
| Weight-for-age | -1.87 | 0.93 | 1,851 | -1.64 | 0.94 | 769 | -2.03 | 0.89 | 1,082 |
| Height-for-age | -1.65 | 1.11 | 1,851 | -1.40 | 1.03 | 769 | -1.83 | 1.13 | 1,082 |
| Weight-for-age (age 1) | -1.54 | 1.12 | 1,841 | -1.27 | 1.07 | 770 | -1.73 | 1.11 | 1,071 |
| <i>Social Measures</i> | | | | | | | | | |
| Activities with children | 3.29 | 1.10 | 1,854 | 3.20 | 1.15 | 772 | 3.35 | 1.06 | 1,082 |
| Played with friends | 0.55 | 0.50 | 1,854 | 0.47 | 0.50 | 772 | 0.60 | 0.49 | 1,082 |

Source: Young Lives Study, India, younger cohort. Health measures standardized against international reference population.

2.5. EMPIRICAL RESULTS

In this section, I begin by evaluating the fit of the model to the empirical data and documenting the role of the unobserved endowments on selection into private school. I then provide estimates of the effects of endowments on outcomes as well as the importance of

the intergenerational transmission of psychosocial traits from mother to child. Finally, I define and present the estimated treatment effects of private school. I also discuss how the treatment effects vary over the distribution of endowments, maternal psychosocial traits, and other observed characteristics.

2.5.1 Fit of the Model and the Role of Endowments

The MCMC estimation provides a posterior distribution of model parameters I use to simulate counterfactual choices and outcomes in order to evaluate the fit of the model and explore the role of the unobserved endowments on selection into private school. Specifically, I draw with replacement N individuals from the sample, and for each make K draws from the joint posterior distribution of parameters.¹⁴ I then simulate the probability of choosing private school for each individual according to:

$$\hat{P}_i = \frac{1}{K} \sum_{k=1}^K \mathbf{1} [\gamma_k Z_i + \lambda_{P,k} \theta_{i,k} + \epsilon_{Pi} > 0],$$

where $\mathbf{1}[\cdot]$ is the indicator function and ϵ is drawn from the standard normal distribution. Notice that unobserved factors for each individual are also drawn from posterior estimates and thus vary over the K draws similar to the other parameters. Outcomes conditional on attending a government-run or private school ($t = 0, 1$) are simulated as:

$$\hat{Y}_i^t = \frac{1}{K} \sum_{k=1}^K (\beta_k^t X_i + \lambda_{Y,k}^t \theta_{i,k} + \epsilon_{Yi}^t)$$

where ϵ is again drawn from a standard normal distribution. Combining results for schooling choice and possible outcomes yields the expected observed outcome for each individual:

$$\hat{Y}_i = \hat{P}_i \hat{Y}_i^1 + (1 - \hat{P}_i) \hat{Y}_i^0.$$

¹⁴ N is the same size as the actual data sample. $K = 10$.

Using the simulated predictions, I next evaluate the fit of the model to the data. A comparison of the simulated and actual mean of private school enrollment is shown in Table 2.4. Note that the model passes a goodness of fit test along the school choice dimension. Comparing predicted psychosocial outcomes to the data requires placing the simulated continuous outcomes back into discrete bins based on the cut-points produced during the MCMC estimation. Figure 2.1 shows the discretized simulated outcomes match the actual data quite well, and the model again passes tests of goodness of fit.

Table 2.4: Model Fit: School Choice

| | Actual | Simulated | Difference | <i>p</i> -value |
|----------------------------|--------|-----------|------------|-----------------|
| Private School ($P = 1$) | 0.417 | 0.420 | -0.003 | 0.747 |

Reported *p*-value from chi-squared goodness of fit test (Null Hypothesis: *Simulated=Actual*).

I next turn to the role of cognitive, health, and social endowments on selection into private school. Figure 2.2 plots the simulated marginal distribution of the latent endowments conditional on type of school attended. There is visible sorting of children with higher cognitive and health endowments into private schools, which is confirmed by Kolmogorov-Smirnov tests. Conversely, there is sorting of children with lower social endowments into private school.¹⁵ This highlights the importance of controlling for latent child endowments in addition to family background when analyzing private school effects.

In order to shed further light on the impact of the unobserved endowments, I compute the change in private school enrollment probability and expected outcomes for exogenous movements along the endowment distributions. I begin with estimating the probability of enrolling in private school for each percentile of a given endowment by integrating out observable covariates and fixing the other endowments at their overall mean.¹⁶ As shown in Figure 2.3, moving along the cognitive and health distributions is associated with an increase

¹⁵The unconditional correlation between cognitive and health endowments is 0.252, between cognitive and social -0.064, and between social and health 0.031.

¹⁶Empirically, for each percentile p , I estimate $E[P | \theta^p] \approx \frac{1}{NK} \sum_{k=1}^K \sum_{i=1}^N \mathbf{1}[\gamma_k Z_i + \lambda_{P,k} \theta_k^p > 0]$. The endowment vector percentile and means are computed for each of the K parameter draws.

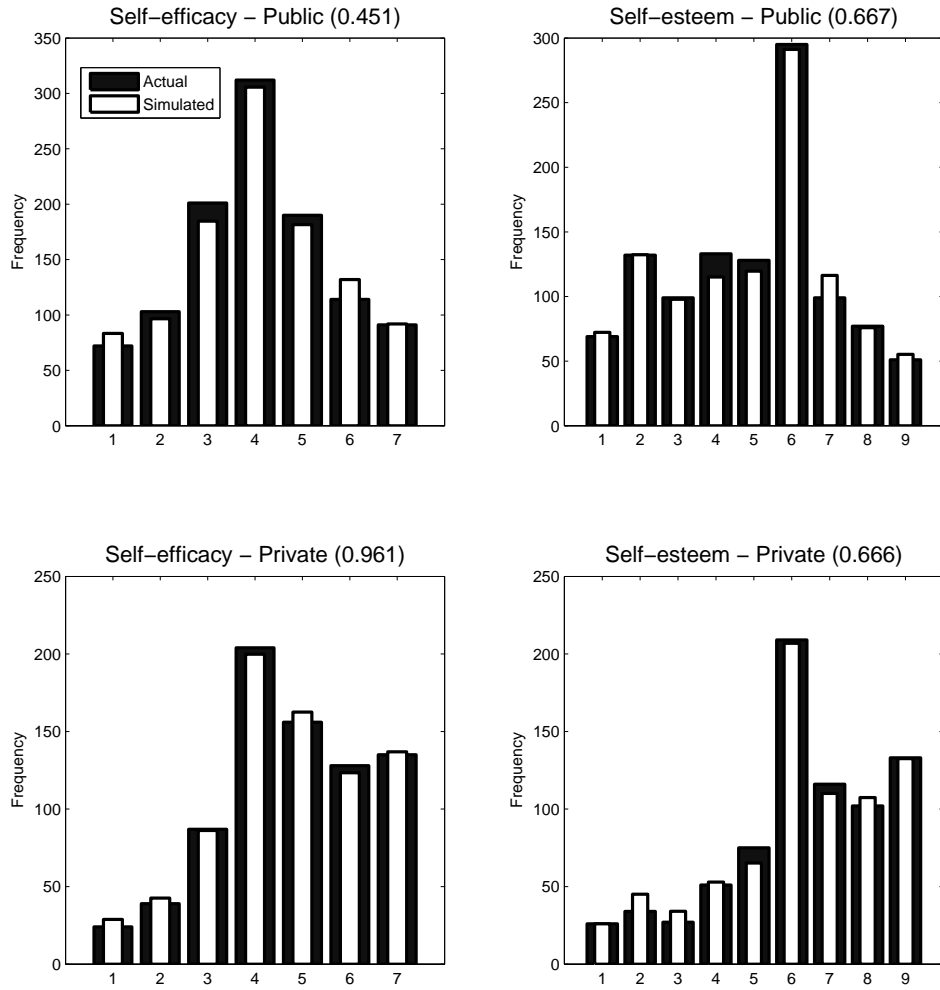


Figure 2.1: Goodness of Fit: Psychosocial Outcomes. Actual data from Young Lives Study, India, younger cohort. Simulations from the posterior estimates of the model. Reported p -value from chi-squared goodness of fit test in parentheses (Null Hypothesis: *Simulated=Actual*).

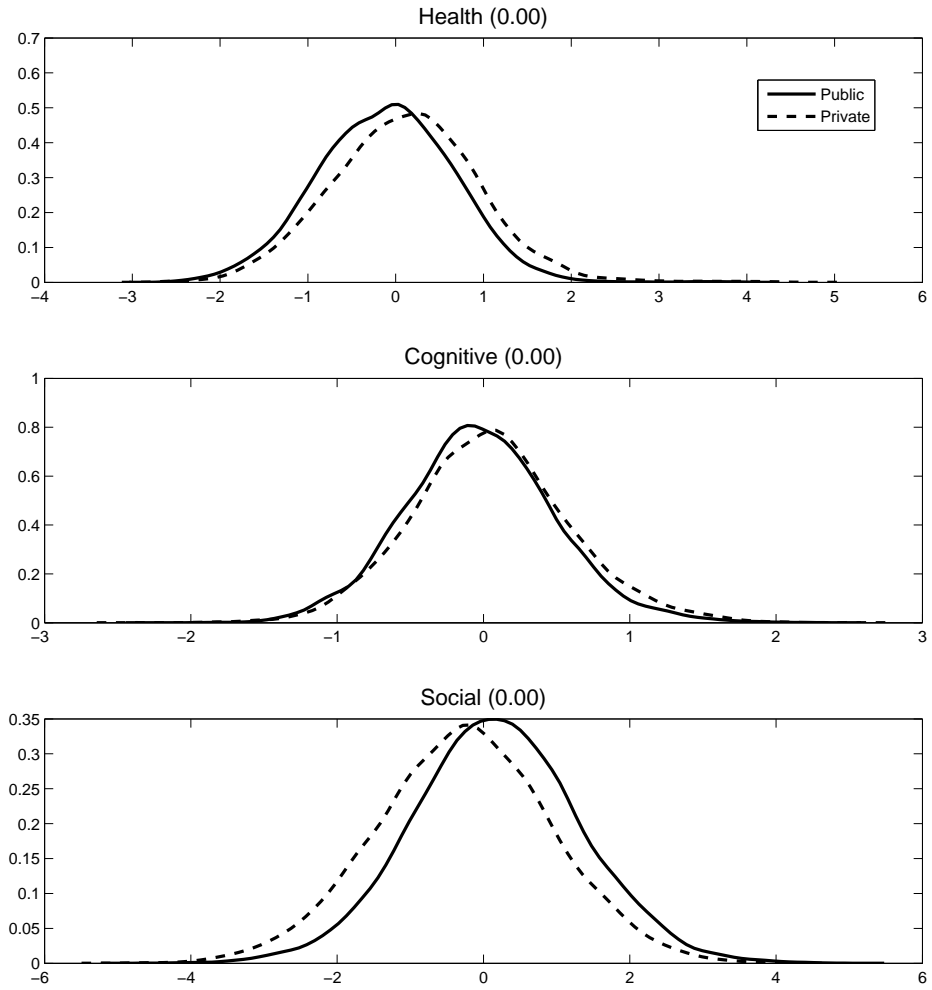


Figure 2.2: Marginal Distribution of Endowments by School Type. Simulations from the posterior estimates of the model. Reported p -value from two-sample Kolmogorov-Smirnov test in parentheses. Null hypothesis that the two distributions are the same.

in the probability of choosing private school. For example, moving from the 20th to 80th percentile of the cognitive or health endowments increases a child's probability of attending private school by approximately 2.0% and 10.4%, respectively. Conversely, an analogous percentile change along the social distribution decreases a child's probability of attending by approximately 18.8%. These results again demonstrate the selection that occurs based on unobserved child traits.

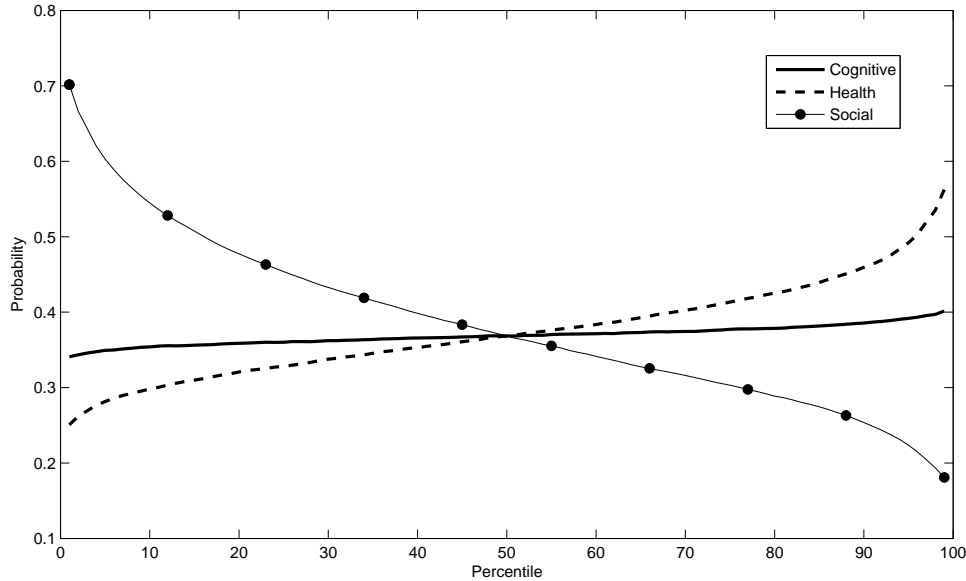


Figure 2.3: Effects of Endowments on Probability of Private School. Simulations from the posterior estimates of the model.

Finally, Figure 2.4 plots the predicted changes in expected unconditional outcomes from exogenous movements along each endowment distribution, again holding the other endowments at their means.¹⁷ Here, I have normalized the expected value at the first percentile to zero to compare the relative effect of moving along a given factor distribution. I have also standardized outcomes to have mean zero and variance one so that effect sizes can be interpreted as standard deviation changes. Comparing endowment effects, an exogenous

¹⁷Empirically I estimate $E[Y^j | \theta^p] \approx \frac{1}{NK} \sum_{k=1}^K \sum_{i=1}^N (\beta_k^j X_i + \lambda_{Y,k}^j \theta_k^p)$ for $(j = 0, 1)$ and then weight by private school probability estimates to predict unconditional outcomes. All other results reported by percentile are estimated analogously.

increase in cognitive skill has the strongest effect on both psychosocial outcomes, followed by an increase in social skill. For example, moving from the 20th to 80th percentile of the cognitive distribution increases a child's self-efficacy and self-esteem by about 0.15 and 0.25 standard deviations, respectively. Analogous estimates for social skill are about 0.10 and 0.15 standard deviations. Moving up the health distribution has a smaller effect on self-efficacy and a negligible effect on self-esteem. Note that expected outcomes are not conditional on private school choice; thus total endowment effects are capturing both direct effects and any expected gains that occur from the increased probability of attending private school.

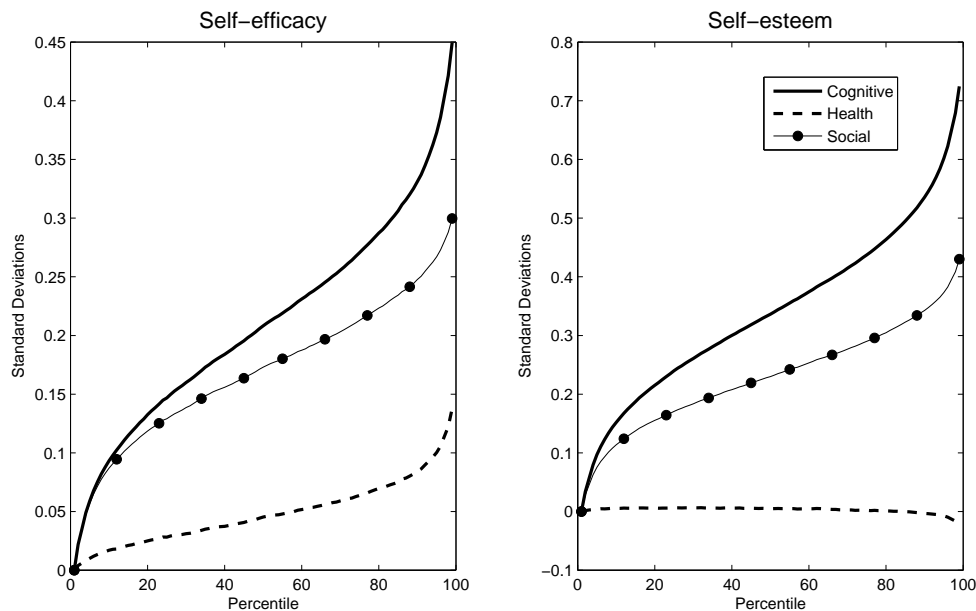


Figure 2.4: Total Effects of Endowments. Simulations from the posterior estimates of the model.

2.5.2 The Intergenerational Transmission of Psychosocial Traits

The inclusion of parental psychosocial measures in the YLS data allows for the unique opportunity to examine if and how such competencies are transmitted from one generation to the next. In order to investigate the intergenerational transmission of noncognitive traits in the sample, I estimate the effects of maternal self-efficacy and self-esteem on private school

enrollment and child outcomes. I follow a similar procedure as estimating endowment effects in the previous section except that other observed covariates are held at their overall mean while the simulated endowments are integrated out of the expected values.¹⁸

The total effects of moving along the maternal psychosocial competency distributions on child outcomes are shown in Figure 2.5. Maternal competencies have large effects on child self-efficacy; moving an otherwise average child from the 20th to 80th percentile of the maternal self-efficacy and self-esteem distributions increases the outcome by 0.34 and 0.26 standard deviations, respectively. There is an even stronger transmission between mother and child self-esteem, though the effect of maternal self-efficacy on child self-esteem is considerably weaker.

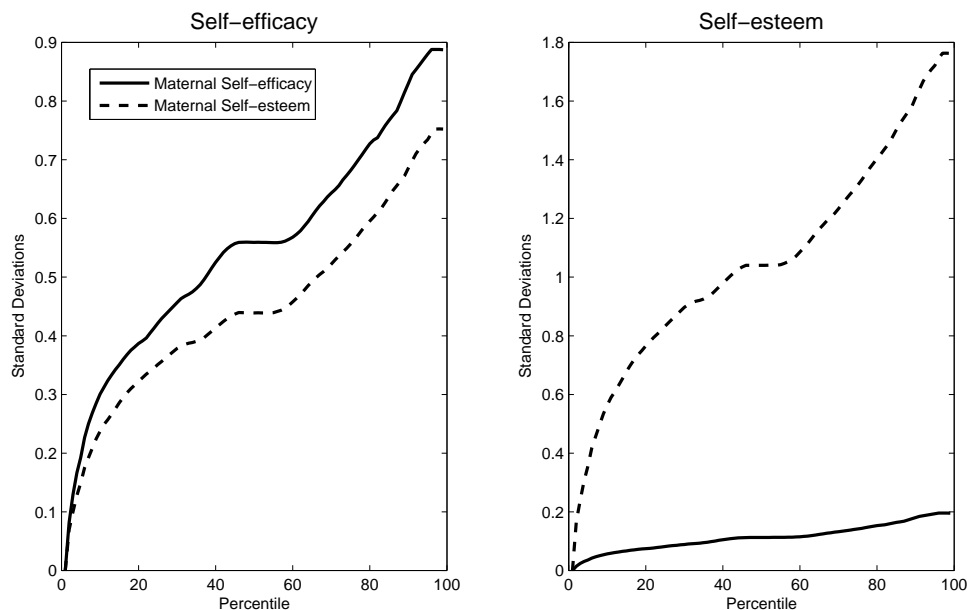


Figure 2.5: Total Effects of Maternal Competencies. Simulations from the posterior estimates of the model.

Of considerable interest to economists and policymakers alike is understanding not only the magnitude of intergenerational transmission of noncognitive traits, but also the underlying mechanisms at work. The model identified in this chapter allows for examination of one

¹⁸Recall that endowments and observed covariates are independent, but covariates themselves may be correlated.

possible channel—private school choice. Figure 2.6 shows that maternal self-efficacy and self-esteem have positive effects on the probability of enrolling in private school. For example, moving an otherwise average child from the 20th to 80th percentile of maternal self-efficacy increases their probability of attending private school by approximately 12%. This suggests that one channel through which psychosocial competencies may be transmitted from mother to child is through the choice of sending the child to private school. In order to compare the importance of this channel relative to the residual “direct” effect, I decompose the total effects of maternal capacities on child outcomes through different channels according to:

$$\begin{aligned} \overbrace{\frac{\partial E[Y | X, \theta]}{\partial X^*}}^{\text{Total Effect}} &= \overbrace{\sum_{t=0}^1 E[Y | X, \theta, P = t] \times \frac{\partial \text{Prob}[P = t | X, \theta]}{\partial X^*}}^{\text{Effect through school choice}} \\ &\quad + \underbrace{\sum_{t=0}^1 \text{Prob}[P = t | X, \theta] \times \frac{\partial E[Y | X, \theta, P = t]}{\partial X^*}}_{\text{Residual "direct" effect}}, \end{aligned}$$

where X^* is maternal self-efficacy or self-esteem. The effect through school choice reflects the expected gain in child outcomes associated with the increased probability of attending private school. The residual “direct” effect is most likely a combination of other behavioral mechanisms and/or genetic transmission of noncognitive traits; the further exploration of which is outside the scope of the current model. In practice, I decompose the simulated total effects of moving an otherwise average child from the 20th to 80th percentile of the maternal competency distributions using finite differencing.¹⁹

The results of the decomposition exercise are presented in Table 2.5. Overall, results suggest that private school choice is a meaningful channel through which maternal self-efficacy influences a child’s psychosocial development. For example, in the exercise, 9% of the effect of maternal self-efficacy on child self-efficacy passes through private school choice. The pass-through percentage from maternal self-efficacy to child self-esteem is over 40%, though recall that the overall magnitude of this effect is relatively small. In contrast, the magnitude

¹⁹E.g. $\frac{\partial \text{Prob}[P=1|X,\theta]}{\partial X^*} \approx \frac{\text{Prob}[P=1|X,\theta]}{X_{80}^*} - \frac{\text{Prob}[P=1|X,\theta]}{X_{20}^*}$

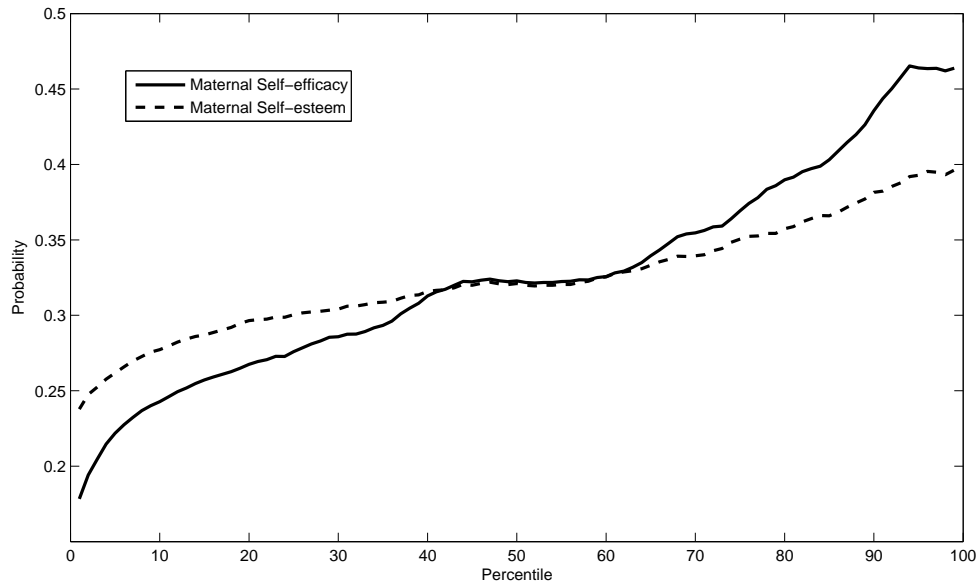


Figure 2.6: Effects of Maternal Competencies on Probability of Private School. Simulations from the posterior estimates of the model.

of the school choice channel for maternal self-esteem effects are significantly smaller—less than 3% for both outcome measures. This highlights that differing transmission mechanisms are at play for alternate facets of psychosocial development.

Table 2.5: Decomposition of the Effects of Maternal Capacities on Child Outcomes

| | Maternal Self-efficacy | | | Maternal Self-esteem | | |
|---------------------|------------------------|--------|-------|----------------------|--------|-------|
| | Private School | Direct | Total | Private School | Direct | Total |
| Child Self-efficacy | 0.09 | 0.91 | 1.00 | 0.03 | 0.97 | 1.00 |
| Child Self-esteem | 0.43 | 0.57 | 1.00 | 0.02 | 0.98 | 1.00 |

Note: Estimates based on simulations of movement for an observationally average child from the 20th to the 80th percentile of maternal self-efficacy and self-esteem distributions.

2.5.3 The Treatment Effect of Private School

This section defines and provides estimates of the causal effect of private school on childhood measures of self-efficacy and self-esteem. Because counterfactual outcomes can be estimated for all individuals in the population regardless of whether they attended private school or

not, the distribution of person-specific treatment effects can be estimated. This highlights one of the main benefits of the model—it allows for estimation of different treatment effects over various subsets of the population.

I begin by defining the average effect of selecting a child at random from the population and enrolling them in private school, also known as the average treatment effect (ATE). The formal definition of the ATE of private school on child outcomes is given by:

$$E[Y^1 - Y^0] = \iint E[Y^1 - Y^0 \mid X, \theta] dF_{X, \theta}$$

where Y^t is the potential outcome of attending private school ($t = 1$) or a government-run school ($t = 0$) and $F_{X, \theta}(\cdot)$ denotes the joint distribution of X and θ . In practice, I use draws from the posterior distribution of parameters (including latent endowments) to estimate the ATE as:

$$E[\hat{Y}^1 - \hat{Y}^0] = \frac{1}{NK} \sum_{k=1}^K \sum_{i=1}^N [E[\hat{Y}^1 \mid (X_i, \theta_{i,k}; \beta_k^1, \lambda_{Y,k}^1)] - E[\hat{Y}^0 \mid (X_i, \theta_{i,k}; \beta_k^0, \lambda_{Y,k}^0)]]$$

where \hat{Y}^j are the potential outcomes predicted by the model.²⁰

Second, I estimate the average effect of the treatment on the treated (ATT) and on the not treated (ATNT). The ATT estimates the effect of private school on a child randomly selected from population of children who were actually observed to attend private school. Analogously, the ATNT estimates the effect on a random child who did not attend private school. Formally, the ATT ($t = 1$) and ATNT ($t = 0$) are given by:

$$E[Y^1 - Y^0 \mid P = t] = \iint E[Y^1 - Y^0 \mid X, \theta, P = t] dF_{X, \theta \mid P=t}$$

where $F_{X, \theta \mid P=t}$ denotes the joint distribution of X and θ given private school choice. Empirically, I again use parameter draws from the posterior distribution to estimate the effects

²⁰The N individuals are the actual sample and $K = 1,000$.

as:

$$E[\hat{Y}^1 - \hat{Y}^0 \mid P = t] = \frac{1}{N_t K} \sum_{k=1}^K \sum_{i:P=t}^{N_t} [E[\hat{Y}^1 \mid (X_i, \theta_{i,k}; \beta_k^1, \lambda_{Y,k}^1)] - E[\hat{Y}^0 \mid (X_i, \theta_{i,k}; \beta_k^0, \lambda_{Y,k}^0)]]$$

where N_t denotes the number of individuals enrolled in private schools ($t = 1$) and government-run schools ($t = 0$).

The estimated treatment effects are reported in Table 2.6. Outcomes have again been standardized so that effect sizes can be interpreted as standard deviation changes. The average treatment effect of attending private school between age five and eight is an increase in self-efficacy and self-esteem measures of 0.15 and 0.28 standard deviations, respectively. These results are within the range of credible estimates of private school effects on cognitive outcomes in India. For example, Muralidharan and Sundararaman (2015) find that private school enrollment increases cognitive test scores an average of 0.23 standard deviations over four years—ranging from no effect to 0.9 standard deviation depending on the school subject tested. This suggests that private school in India may play at least as significant a role in early psychosocial as in cognitive development.

Table 2.6: Treatment Effects of Private School

| | ATE | ATT | ATNT |
|---------------|------------------|------------------|------------------|
| Self-efficacy | 0.152 (0.063) | 0.125 (0.079) | 0.172 (0.072) |
| Self-esteem | 0.278 (0.056) | 0.315 (0.074) | 0.251 (0.065) |

Note: Standard errors in parentheses.

When moving beyond the ATE, the estimated treatment effects differ based on observed school choice, though differently for the two outcomes (see Table 2.6 and Figure 2.7). Effects on self-efficacy are stronger among children that did *not* attend private school, while the opposite is true for self-esteem. These results are consistent with a limited role of self-efficacy gains in determining if parents send their child to private school—at least relative

to other determining factors. However, it does leave scope for some sorting based on gains to self-esteem.

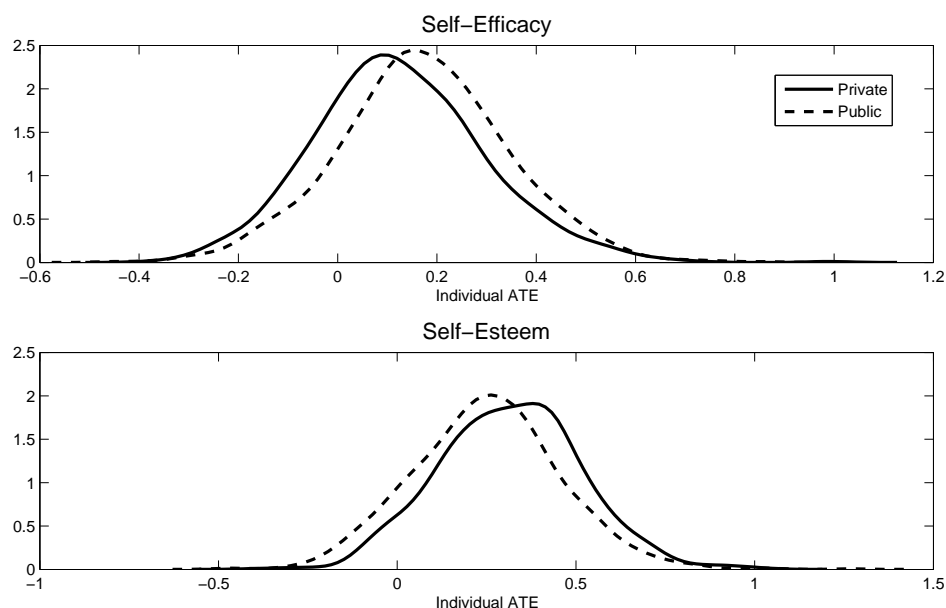


Figure 2.7: Distribution of Treatment Effect by School Type

Finally, I decompose the difference in simulated outcomes to gain a better understanding of how treatment effects are driving the observed differences between private and public school. Specifically, the observed difference in noncognitive outcomes presented in Table 2.1 is due to a combination of the ATT and selection bias. Table 2.7 presents the decomposition for each outcome into these two sources. There is stark contrast in this exercise between the noncognitive measures. Selection accounts for about 54% of the observed difference in self-esteem, while the remaining 46% is attributed to the treatment. In contrast, 72% of the observed difference in self-efficacy is due to selection, while the treatment accounts for only 28%. The distribution of treatment effects across family and child characteristics presented in the next section help shed light on the source of these differences.

Table 2.7: Decomposition of Observed Differences in Outcomes

| | Observed Diff | ATT | % ATT | Selection | % Selection |
|---------------|---------------|-------|-------|-----------|-------------|
| Self-efficacy | 0.449 | 0.125 | 27.8 | 0.324 | 72.2 |
| Self-esteem | 0.685 | 0.315 | 46.0 | 0.370 | 54.0 |

Note: Observed difference simulated from the model estimates.

2.5.4 Treatment Effect Heterogeneity

Understanding which children may or may not benefit from private schooling has clear implications for parents and policymakers alike. In this section, I provide estimates of the heterogeneity of treatment effects along a number of household and child characteristics. I begin by estimating the distribution of effects across a number of dichotomous observables—gender, urbanization, and caste. Results are presented in Table 2.8 as well as Figures 2.8-2.10.²¹ While there is little difference between genders on average self-efficacy gains, males have higher potential gains on self-esteem. The average treatment effect on self-esteem was also stronger for children living in urban areas. In contrast, rural students have higher potential gains on self-efficacy from attending private school, though estimates are less precise. Finally, while there is little difference between castes on average gains to self-esteem, children from Scheduled Castes or Scheduled Tribes have significantly higher potential gains on self-efficacy. This has direct implications for the recently passed Right to Education Act, which effectively moves children from these lower castes into private school. The effects of this policy on child psychosocial outcomes is explored further in the later counterfactual analysis.

I next document the heterogeneity of treatment effects along the unobserved child endowments. Specifically, Figure 2.11 presents the average treatment effect for each percentile of a given endowment after integrating out observable covariates and fixing the other endowments at their overall mean. The effect of the treatment on self-efficacy is stronger at the top of each of the endowment distributions, suggesting that endowments and private

²¹Effects are computed analogously to ATT and ATNT.

Table 2.8: Average Treatment Effect by Select Observables

| | Male | Female | Urban | Rural | SC/ST | OC |
|---------------|------------------|------------------|------------------|------------------|------------------|------------------|
| Self-efficacy | 0.138 (0.075) | 0.169 (0.082) | 0.111 (0.125) | 0.166 (0.069) | 0.214 (0.100) | 0.124 (0.071) |
| Self-esteem | 0.349 (0.064) | 0.197 (0.076) | 0.377 (0.116) | 0.246 (0.062) | 0.276 (0.091) | 0.278 (0.062) |

Note: Standard errors in parentheses. SC/ST = Scheduled Caste or Tribe, OC = other caste.

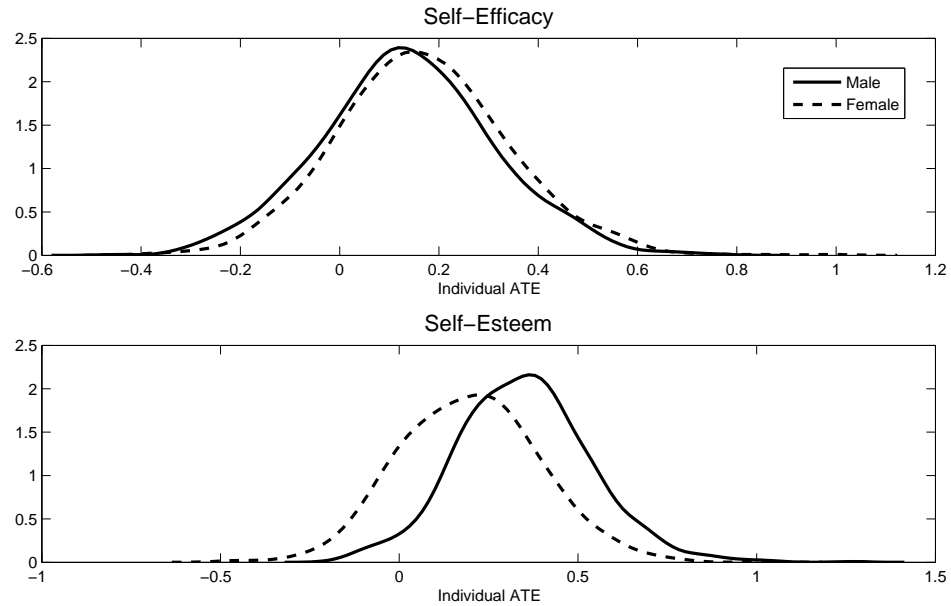


Figure 2.8: Distribution of Treatment Effect by Gender

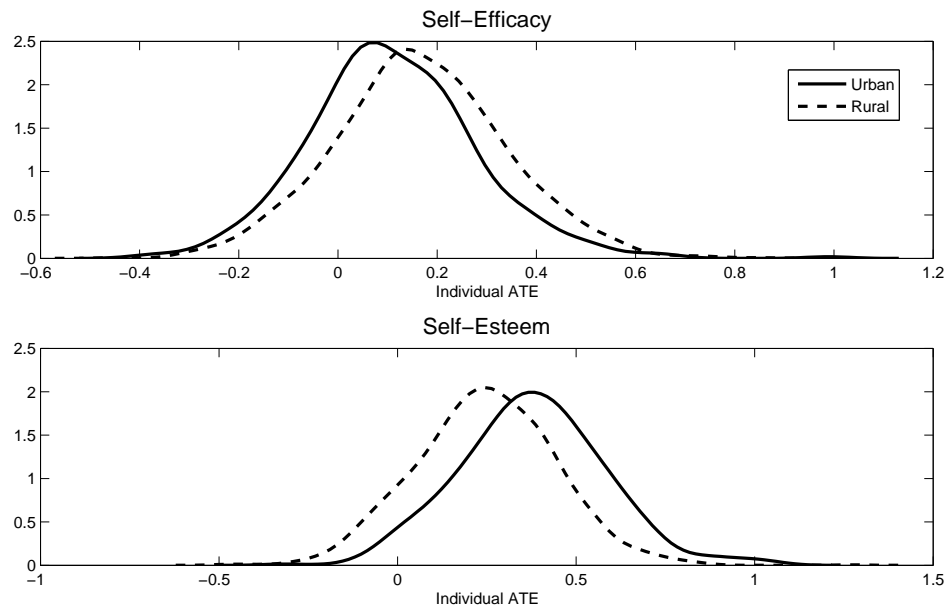


Figure 2.9: Distribution of Treatment Effect by Urban Status

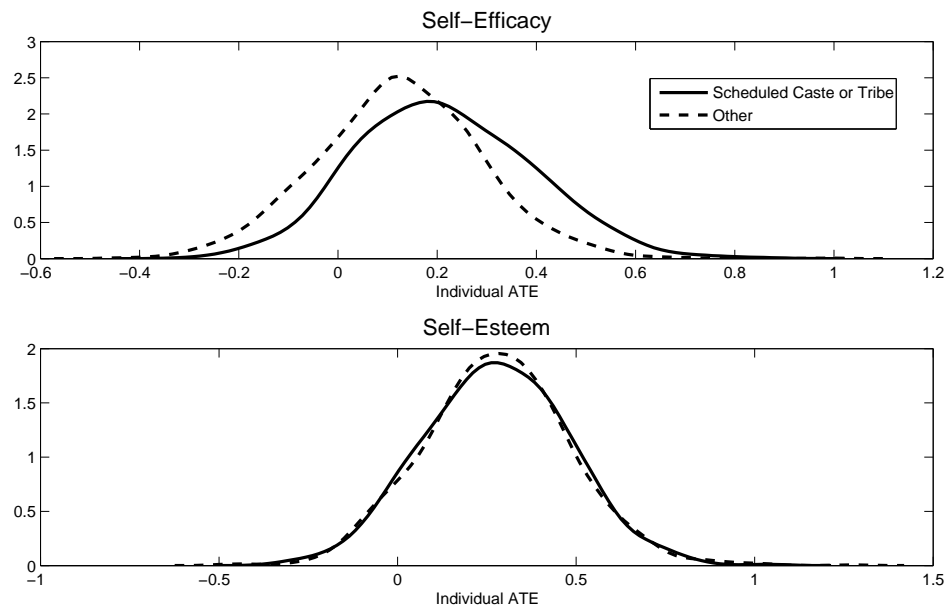


Figure 2.10: Distribution of Treatment Effect by Caste

school are complements in the production of the outcome. In other words, children with higher cognitive, health, or social endowments can expect higher gains to self-efficacy from attending private school. A similar relationship is found between self-esteem and cognitive and social endowments. Conversely, effects on self-esteem are strongest at the bottom of the health distribution. This suggests that to some extent private school may serve as a substitute for health in the development of a child's self-esteem.

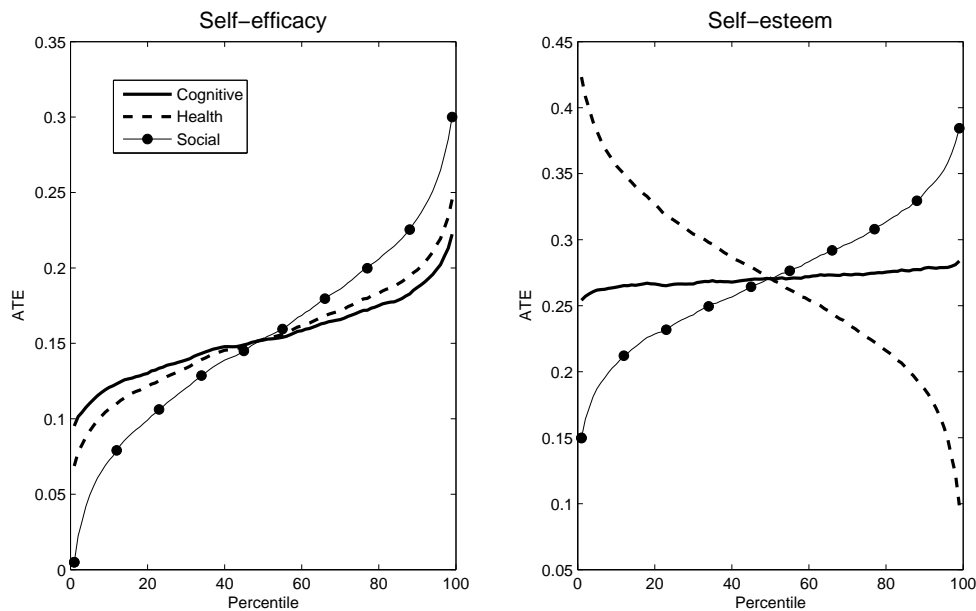


Figure 2.11: Treatment Effect Heterogeneity: Endowments

Turning now to the role of parental psychosocial traits in determining treatment effects, I examine the heterogeneity of treatment along the maternal self-efficacy and self-esteem distributions. Again, results are presented for each percentile of a given characteristic after integrating out endowments and holding other covariates at their respective means. As shown in Figure 2.12, maternal self-esteem and private schooling play complementarity roles in development of the child outcomes. In contrast, maternal self-efficacy does not impact treatment effects on child self-esteem, but has a very strong influence on child self-efficacy effects. For example, moving from the 20th to 80th percentile of the maternal self-efficacy distribution *decreases* the average treatment effect on child self-efficacy by about 0.24 standard

deviations. This suggests that private school may serve as a substitute for the acquisition of self-efficacy through parental transmission.

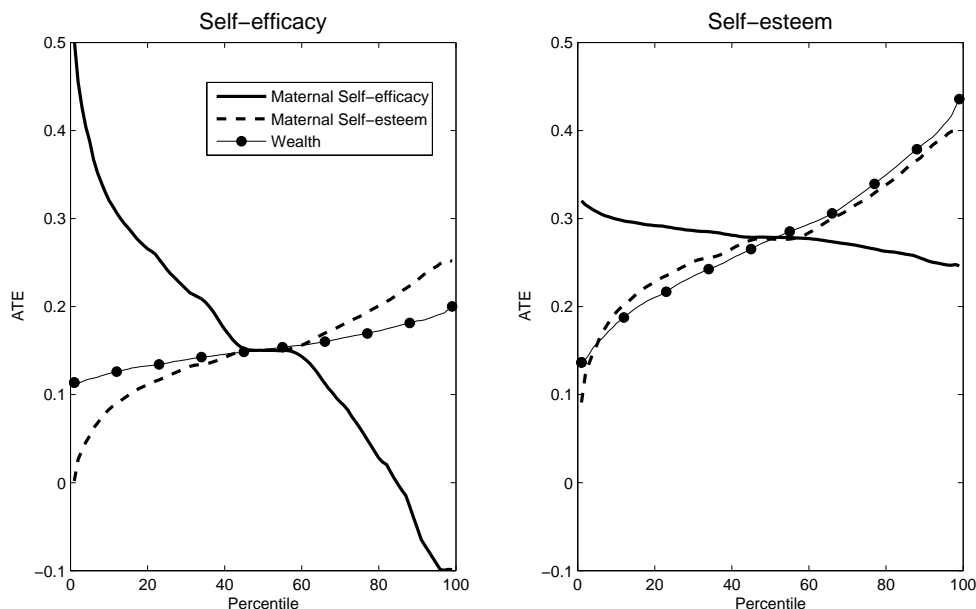


Figure 2.12: Treatment Effect Heterogeneity: Household Characteristics

The final source of heterogeneity I examine is household wealth. The analysis presented thus far have excluded discussion on how variation in the quality of private schools could be influencing results. As an example, the complementarity between cognitive endowment and private school could be driven by children with higher cognitive endowments being sent to higher quality private schools. As an arguably more direct examination of this school quality influence, Figure 2.12 presents the heterogeneity of treatment effects along the household wealth distribution. Results show that wealth and private school are complements in the production of each of the outcomes; though the relationship is stronger for self-esteem. This is consistent with wealthier parents sending children to higher quality private schools resulting in significantly higher noncognitive returns; however there could be other mechanisms at work as well. Note that this does not invalidate the findings presented thus far as wealth and other covariates serve to control for variation in school quality in all model estimates. However, heterogeneity along wealth and other dimensions does caution that the main results

should be viewed as average effects as private school quality likely plays a role in determining psychosocial gains from attendance.

In light of the observations on treatment effect heterogeneity, I now return briefly to the results presented in the previous section. First note that selection into private school is positively influenced by child endowments, maternal psychosocial traits, wealth, being male, coming from a higher caste, and living in an urban area. Almost all of these characteristics are also associated with higher treatment effects on child self-esteem.²² In contrast, for self-efficacy, it is children with low maternal self-efficacy, coming from a lower caste, and living in a rural area that have the most to gain from the treatment. However, these are the very children who are less likely to be enrolled in private school. These observations help explain the results presented in Table 2.6 and decomposed in Table 2.7. Namely, the ATT is higher than the ATNT for self-esteem, while the opposite is true for self-efficacy. These findings also have a clear influence on the results of the counterfactual analysis presented in the next section.

2.6. THE RIGHT TO EDUCATION ACT: A COUNTERFACTUAL ANALYSIS

India passed the Right to Education Act (RTE) in 2009, making education a fundamental right for every child aged 6 to 14. As part of the law, private schools are required to reserve 25% of capacity for enrollment of disadvantaged populations—predominately children from Scheduled Castes or Scheduled Tribes.²³ The cost of attendance is to be reimbursed to private schools by the state. This policy of caste-based reservation in private schools stems from the perceived difference in quality between school types. Policymakers believe the gap

²²Caste and health endowment are the only exceptions.

²³Orphans, HIV affected, disabled, and the very poor may also qualify as disadvantaged.

between disadvantaged children and their more fortunate counterparts is worsened by the disparity in private school enrollment.

In order to shed light on how the RTE may effect the psychosocial development of different populations of children in India, I conduct a counterfactual simulation roughly consistent with the policy change. Specifically, I estimate the effect of moving the required number of disadvantaged children from government-run schools into private schools to reach the 25% threshold for the sample population. These children are not, however, selected at random from the pool of qualified candidates. Instead I move the children with the highest net utility from attending private school but who would choose not to enroll without the simulated policy change. Intuitively, this assumes that it is the disadvantaged children closest to the utility threshold for enrollment that will be pushed to attend private school by the policy (i.e. $\max \{P^* < 0\}$).

I conduct the analysis under two alternate assumptions about the implementation of the policy. First, I assume private school capacity is fully flexible and increases to allow the required enrollment of disadvantaged students without the displacement of others. Second, I assume the overall capacity of private schools remains unchanged, and the addition of disadvantaged children to obtain the 25% threshold results in the direct displacement of children who would otherwise attend private school. Displaced children are also not selected at random, but are identified as having the lowest net utility from private school of those who would choose to enroll without the policy change and are not classified as disadvantaged.

The results of the counterfactual policy experiments are presented in Table 2.9. The first column shows the effects on the disadvantaged children moved into private school due to the policy change under the fixed capacity scenario. On average, these children experienced an increase in self-efficacy of nearly 0.21 standard deviations. Note that this is considerably higher than the estimated effect on a child drawn at random from the entire population (i.e. the ATE of Table 2.6). However, the expected increase in self-esteem of 0.27 is quite similar to that of a randomly drawn child. In contrast to the affected disadvantaged children, those

displaced from private school by the policy experience lower expected outcomes. This highlights a significant potential cost of the proposed policy. However, notice that the positive point estimates for the disadvantaged children are larger in magnitude than the negative effects on displaced children for self-efficacy. This is due to the heterogeneity of treatment effects discussed in the previous section. Finally, the last column shows the results assuming flexible private school capacity and no displacement. The average effects on disadvantaged children moved into private school are very similar to the fixed capacity scenario. However, the number of disadvantaged children affected by the policy change increases from 2.1% to 3.3% of the population.

Table 2.9: Counterfactual Results

| | Fixed Capacity | | Flexible Capacity |
|---------------------|------------------|-------------------|-------------------|
| | Treated | Displaced | |
| Self-efficacy (ATE) | 0.205 (0.109) | -0.128 (0.084) | 0.205 (0.104) |
| Self-esteem (ATE) | 0.271 (0.103) | -0.274 (0.078) | 0.270 (0.097) |
| % of Population | 2.1 (0.69) | 2.1 (0.69) | 3.3 (0.76) |

Note: Standard errors in parentheses. Treated refers to the children moved into private school due to the policy change.

When interpreting the counterfactual results in terms of the RTE, several caveats warrant further discussion. First, the RTE requires each private school to reserve 25% of capacity for disadvantaged children, not 25% of capacity as a group. In reality, individual private schools are at various levels of compliance, with some well over the required 25% threshold at the baseline. This implies the estimated quantity of disadvantaged students affected by the policy is likely underestimated in the above experiment. Furthermore, the individual treatment effects are likely more nuanced due the patterns of compliance across private schools of varying quality. Second, the experiments above do not account for possible spillover effects of the changing enrollment patterns. For example, a large swell of disadvantaged students in

private schools could conceivably have negative consequences on the psychosocial development of those already enrolled. I will note, however, that Muralidharan and Sundararaman (2015) find no evidence of negative spillover effects on cognitive outcomes in their randomized school voucher trial. Despite these caveats, the counterfactual analysis provides valuable insights into the patterns and potential costs that may be expected to arise from the continued implementation of the RTE.

2.7. CONCLUSIONS

This chapter provides novel evidence of the influence of school context on early noncognitive development of children in India. I find that the effects of private schooling on child self-efficacy and self-esteem are at least as large as previously estimated effects on many cognitive test scores. Furthermore, I find effects differ across important family characteristics as well as unobserved child endowments. I also uncover differences in how private schooling and other individual characteristics affect alternate measures of psychosocial development (i.e. self-efficacy versus self-esteem). This highlights the importance of recognizing the multidimensional nature of what is often coined “noncognitive skill” in emerging economic research.

Understanding the effects of Indian private schools on self-efficacy and self-esteem can begin to delineate which factors of schooling are most important for a child’s developmental health and help target educational policies most effectively. This chapter takes an important step towards understanding the mechanisms linking educational context to the development of noncognitive traits in young children in the developing world. In future work, I plan to further explore what dimensions of private schooling—and education more broadly—are most impactful on early social and emotional development.

3. CHILDHOOD HEALTH AND PRENATAL EXPOSURE TO SEASONAL FOOD SCARCITY IN ETHIOPIA¹

3.1. INTRODUCTION

An estimated 805 million people globally—roughly one out of every nine—still suffer from chronic undernourishment (FAO 2014). In many of the poorest developing countries, including Ethiopia, estimates are more than one out of every three. Despite a long history of attempts to address food security, an estimated 40% of households in Ethiopia are still classified as food energy deficient by the World Food Programme (2014). A heavy reliance on small-scale rainfed agriculture combined with highly localized agricultural markets make Ethiopia’s erratic climatic conditions a significant source of food uncertainty. Moreover, even in years of fairly typical seasonal patterns of cultivation, lack of storage capacity and costly transport can lead to measurable differences in food availability over the agricultural cycle (FAO 2004; WFP 2014). While seasonal effects on nutritional intake may be mild in comparison to more extreme weather phenomenon (i.e. drought, flood, monsoon), they could

¹The data used in this chapter come from Young Lives, a 15-year study of the changing nature of childhood poverty in Ethiopia, India (Andhra Pradesh), Peru and Vietnam (www.younglives.org.uk). Young Lives is core-funded by UK aid from the Department for International Development (DFID) and co-funded from 2010 to 2014 by the Netherlands Ministry of Foreign Affairs. The views expressed here are those of the author. They are not necessarily those of Young Lives, the University of Oxford, DFID or other funders.

still have a substantial impact among vulnerable groups of the population. In this chapter I focus on one such group—children in the fetal stages of development.

There is growing support in the biomedical literature for the hypothesis that poor maternal nutrition during pregnancy can lead to permanent fetal adaptations that affect health throughout a child’s life (Gluckman and Hanson 2005). This “fetal origins” hypothesis has recently garnered interest among economists, who have attempted to establish and quantify the casual impact of such a mechanism.² These studies have often used uncommon and arguably exogenous events such as famine or disease epidemic to identify the causal effects of prenatal nutritional environment. While many of these studies find significant effects from such environmental shocks, less is known about the magnitude of effects due to more normal variations in food availability.

In this chapter, I use a unique longitudinal data set to examine the effects of prenatal exposure to seasonal variations in food scarcity on childhood health in Ethiopia. My exposure measure is derived by combining individual date of birth with survey data collected shortly after birth at the local community level. Importantly, the survey contains explicit data on months when food becomes harder to obtain or more expensive within each community. Identification relies on the assumption that prenatal exposure to reported months of food scarcity, conditional on community and month of birth fixed effects, is uncorrelated with any unobserved determinants of examined child health outcomes. Under this assumption, I am able to identify the impact of in-utero exposure to reported seasonal food scarcity on health outcomes measured at age one, five, and eight for a cohort of Ethiopian children born between May 2001 and May 2002. My main finding is that exposure has a significant negative impact on height by age five that strengthens by age eight. Moreover, effects decrease with household wealth and maternal education and are stronger when exposure occurs during the first trimester of gestation. In contrast, effects on child body mass are only identified at age one and when exposure is concentrated in the second trimester.

²For a review of the literature, see Currie (2009); Almond and Currie (2011); Currie and Almond (2011); Currie and Vogl (2013).

There are many studies of developed countries that have found month or season of birth to be robustly correlated with health outcomes such as birthweight, life expectancy, and height.³ A smaller but growing body of literature has established similar patterns in the developing world. For example, using a sample of Indian children under three years of age, Lokshin and Radyakin (2012) find those born during monsoon months are significantly shorter than children born in fall or winter months. In Gambia, Rayco-Solon et al. (2005) show the incidence of small-for-gestational age was higher among children born at the end of the “hungry season,” while the peak incidence of preterm births paralleled increases in agricultural labor demand and malaria infections. Moore et al. (2004) show that birth during the hungry season resulted in increased infant mortality rates in both Gambia and Bangladesh. The association between season of birth and health outcomes also appears to persist into adulthood in the developing world. For example, McEniry and Palloni (2010) find in-utero exposure to the hungry season was associated with higher probabilities of heart disease and diabetes among a sample of older Puerto Ricans. Researchers have most commonly argued that prevalence of disease, seasonal maternal labor supply, or nutritional intake associated with agricultural output are likely channels through which calendar time of birth may affect health outcomes in the developing world. While much of the season of birth literature is suggestive that the timing of birth in relation to the agricultural cycle is important, it is difficult to disentangle prenatal nutritional effects from exposure to disease or other seasonal factors.

There is a growing body of related economic literature that examines the effects of early exposure to localized weather shocks. These studies have most commonly relied on changes in annual rainfall or ambient temperature patterns as exogenous sources of variation. While this literature arguably implements stronger identification strategies than the season of birth literature, it still has trouble isolating the relevant mechanisms at work. Maccini and Yang (2009), for example, find higher rainfall in early life is associated with better health outcomes

³See, for example, Doblhammer and Vaupel (2001); Kihlborn and Johansson (2004); Tanaka et al. (2007); Strand et al. (2011).

for Indonesian women, but not for men. They attribute the positive association to the influence of rainfall on increased agricultural output and lower food prices. Rocha and Soares (2015), on the other hand, attribute a positive relationship between rainfall and birth outcomes in Brazil to increased access to safe drinking water and consequently lower prevalence of disease. Still other studies find increased rainfall early in life has negative consequences for later outcomes (e.g. Kim 2010; Aguilar and Vicarelli 2011). Such negative correlations are generally attributed to the disease environment, increased maternal labor supply, or a negative impact of excessive rain on agricultural production. In Africa, for example, Kudamatsu et al. (2014) find that increased rainfall can negatively effect infant mortality in regions with epidemic malaria while drought shocks can have a negative impact in arid areas. Similarly for Mexico, Skoufias et al. (2011) find that weather shocks associated with rainfall and temperature can have substantial negative as well as positive effects that vary across geographic regions.

In this chapter, I make strides towards isolating the impact of seasonal changes in prenatal nutrition on health outcomes by using a treatment measure that is both localized and explicitly based on exposure to food scarcity. A complication with using environmental shocks for identification is the presence of multiple channels through which weather changes have been argued to effect health outcomes. Moreover, even when weather patterns are convincing linked to changes in agricultural production, it is not inherently clear how effects travel through the supply chain and ultimately impact food availability and/or prices. To my knowledge, this is the first study to use an explicit measure of food scarcity to circumvent the ambiguity surrounding the use of environmental shocks, such as rainfall or temperature, as instruments for nutritional deprivation. This is also the first study to use a localized instrument to examine how season of gestation impacts later health outcomes. Localization of the measure allows me to control for seasonal trends that occur at the country level but are unrelated to food availability. Finally, beyond improved isolation of prenatal nutritional environment, having health outcomes collected repeatedly over a period of rapid physical

growth adds novel evidence on gestation being a “sensitive” period of development. Specifically, the pattern of effects across time shed light on how easy or hard it is to make up for poor nutrition during early development with increased health inputs at later stages.

Consistent with the view of gestation as a “sensitive” period of development, I find that exposure has a significant negative impact on child height that strengthens as children age. Specifically, I estimate that an additional month of prenatal exposure to reported seasonal food scarcity decreases height by at least 0.31 cm by age five and 0.41 cm by age eight. The magnitude of these effects are similar to a one-third standard deviation decrease in the household wealth index used in my benchmark specification. As an outside comparison, Dercon and Porter (2014) estimate that infant exposure to the 1984 Ethiopian famine decreased height at least 5 cm by early adulthood and was accompanied by an estimated annual income loss of 5%.⁴ I also find that effects decrease monotonically with household wealth and maternal education and are stronger during the first trimester of gestation. In contrast to height, effects on child body mass are statistically significant only at age one for exposure during the second trimester. This is consistent with height and weight being measures of health variation in the long-run and short-run, respectively. I find no evidence that seasonal variation of water quality or maternal labor supply are driving results, suggesting that I am indeed capturing the effects of seasonal changes in nutritional intake on child health outcomes. Likewise, I find no evidence that results are driven by seasonal fertility patterns within local communities or selective mortality of children on the basis of exposure to seasonal food scarcity. Overall, my results highlight that in addition to the effects of severe famine conditions identified in many studies, more typical variation in prenatal nutritional environment can have lasting impacts on health in the developing world.

The remainder of this chapter is presented as follows. Section 3.2 begins by discussing the data and construction of the prenatal exposure measure. Section 3.3 describes the em-

⁴Dercon and Porter (2014) estimated a significant impact only on those aged 12-36 months during the peak of the famine. Effects are not identify for children in-utero, although the authors cannot rule out that potentially severe mortality selection dominates scarring for this group.

pirical strategy, and Section 3.4 presents the results. Section 3.5 assesses potential alternate channels of correlation including seasonal patterns of fertility, water quality, seasonal labor supply, and selective mortality. Finally, Section 3.6 concludes.

3.2. DATA

I use unique data from the Young Lives Study (YLS) to conduct an empirical analysis of the impact of prenatal exposure to relative food scarcity on later child health outcomes in Ethiopia. The YLS conducted surveys for a cohort of 2,000 children born between May 2001 and May 2002 in twenty sites across the country. Currently data is available from three rounds of surveys conducted in 2002, 2006, and 2009—when children were approximately one, five, and eight years old. The study collects detailed information on household and child characteristics, including anthropometric markers such as height and weight. In addition, a community level survey was conducted during the first wave of data collection, when children were 6-18 months old. These data were obtained on a variety of topics through interviews with key community leaders such as government officials, municipal leaders, and village headmen.

3.2.1 A Measure of Prenatal Exposure to Seasonal Food Scarcity

Regional agroecosystems across Ethiopia are quite diverse, particularly in terms of rainfall and elevation. This can result in substantial variations in crop yield patterns across geographic regions. Moreover, agricultural markets in Ethiopia consist primarily of small farmers and traders who produce and sell product in local markets. According to a 2004 report from the Food and Agriculture Organization of the United Nations, “these [local] markets function in relative isolation and grain movements from surplus to deficit areas are constrained by high transport costs due to poor road infrastructure, weak market information

systems, and a quasi monopoly in the transportation sector.” The combination of a limited agricultural market and varied agroclimatic conditions results in meaningful variations in food availability patterns across geographic regions of Ethiopia.

In light of these geographic diversities, I use relevant data collected at the local community level to construct my measure of prenatal exposure to seasonal food scarcity. A community survey was conducted at each of the sites selected to participate in the YLS. While poor and food-poor areas were oversampled by the study, the communities span Ethiopia geographically, and are contained in the regions where almost 97% of the population reside. Specifically, communities were sampled from the capital city of Addis Ababa and the regional states of Amhara, Oromia, Southern Nations, and Tigray.

My exposure measure is constructed on the basis of the following community survey question:

In which months of the year does food become harder to obtain / more expensive?

Data collectors recorded responses to this question by ticking ‘yes’ or ‘no’ for each month of the year. I use the survey responses for 22 of the 23 local communities, with the last community excluded from analysis due to missing food scarcity data. On average, non-missing communities reported just over 4.5 months of relative food scarcity, with a range of three to eight months (see Figure 3.1). Figure 3.2 shows the percent of all communities reporting food scarcity by calendar month. In anticipation of later analysis, data is also reported for the subset of five communities in or around the Ethiopian capital of Addis Ababa. On aggregate, more communities reported relative scarcity from October to January while less reported scarcity from March to June. This corresponds to a reported average increase in food availability following what is considered Ethiopia’s main harvest season, which typically runs from October to February.

The community surveys were conducted during the last few months of 2002, shortly after the youngest children in the cohort of interest were born. While it was not explicitly specified that respondents answer the food scarcity question in relation to the most recent year or

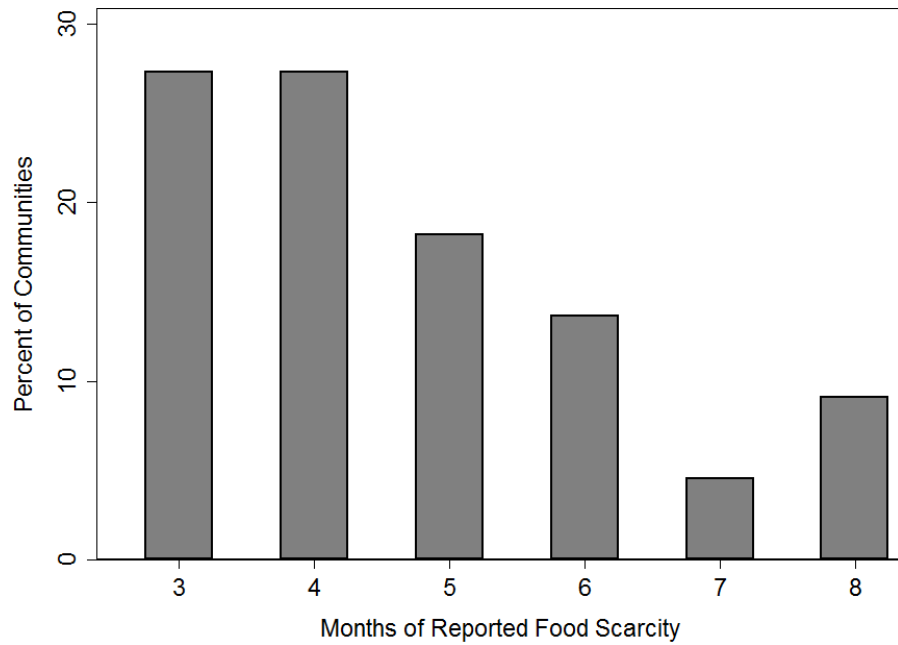


Figure 3.1: Number of Reported Months of Seasonal Food Scarcity. Source: Author's calculations using data from Young Lives Study, Ethiopia.

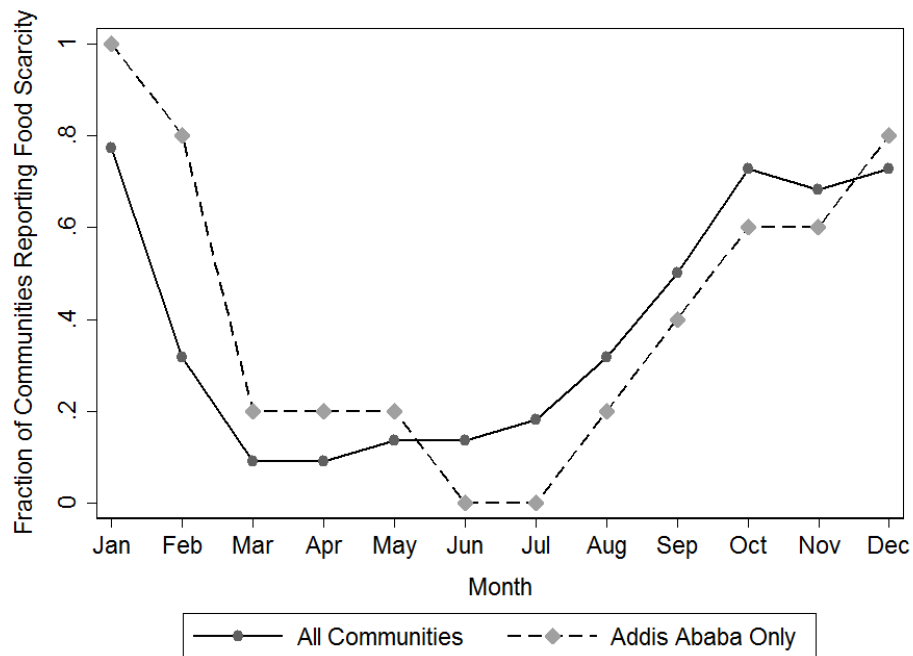


Figure 3.2: Reported Seasonal Food Scarcity by Calendar Month. Source: Author's calculations using data from Young Lives Study, Ethiopia.

years, responses are consistent with available food pricing data. Figure 3.3 shows the monthly price in Addis Ababa of the four major grains harvested in Ethiopia from September 2000 to May 2002. As sample children were born between May 2001 and May 2002, the examined time frame completely spans when the children were in-utero. The observed pattern of prices over the time frame is generally consistent with the data on relative food scarcity reported by the Addis Ababa survey communities shown in Figure 3.2—higher prices from September to February and lower from March to August. Thus, the pricing data provides further evidence that responses from the community survey measure seasonal food scarcity while sample children were in-utero.

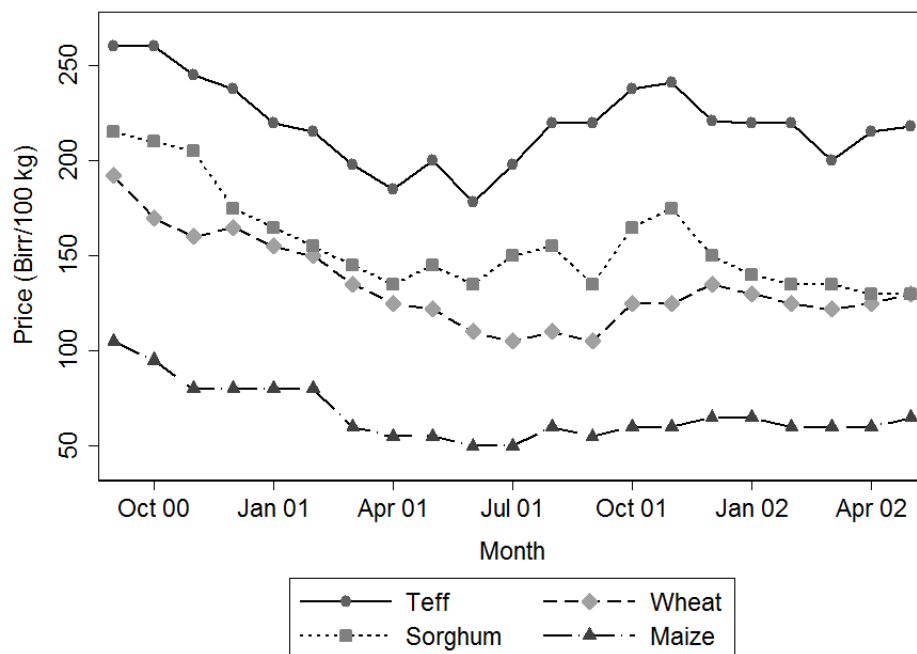


Figure 3.3: Monthly Average Prices of Main Cereals, Addis Ababa. Source: FAO (2004).

I combine the community level food scarcity data with individual date of birth to compute the days of prenatal exposure to relative food scarcity for each child. Conception for a full-term birth is estimated precisely 270 days prior to birth allowing division of the gestational period into three trimesters of 90 days each. For preterm births, gestation length and corresponding exposure days are adjusted based on the reported number of weeks pre-

mature.⁵ The density of the exposure measure is shown in Figure 3.4. On average, children were exposed to an estimated 103 days of reported food scarcity in-utero, with a standard deviation of 49 days.

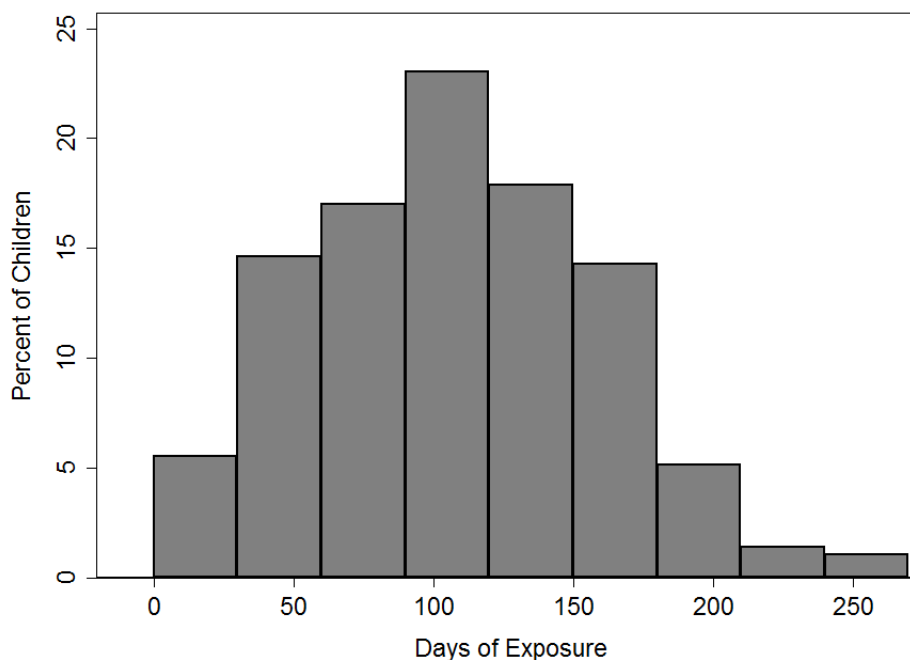


Figure 3.4: Prenatal Days Exposed to Reported Seasonal Food Scarcity. Source: Author’s calculations using data from Young Lives Study, Ethiopia. Histogram with 30 day window for each bin.

3.2.2 Health Outcomes and Other Data

In my empirical analysis I focus on two child health outcomes—height and body mass index.⁶ Height captures a child’s restricted growth potential associated with the chronic or long-term effects of malnourishment. In contrast, body mass is more sensitive to short-term health changes as it captures weight loss associated with acute undernutrition. Outcomes were measured at each round of data collection—when children were approximately one, five, and

⁵8% of all births were reported as preterm. Of these, 75% reported the number of weeks premature. To construct exposure for the remaining 25%, I assign them the median of reported weeks premature (two weeks).

⁶Body mass index is calculated as weight (kg) divided by the square of height (m).

eight years old. Repeated observations during this period of rapid physical development is one of the benefits of the YLS data. Specifically, the pattern of effects across time can shed light on how easy or hard it is to make up for poor nutrition during early development with increased health inputs at later stages. Panel A of Table 3.1 gives descriptive statistics for the outcomes at each age. For ease of interpretation, I express height in centimeters and I standardize the body mass index to have mean zero and variance one. By either measure, malnutrition is quite severe among the sample population of children—stunting and wasting prevalence were both over 20% by age eight.⁷

In addition to prenatal exposure to reported food scarcity, a number of household and child characteristics are used in the empirical analysis to help control for demographic and socioeconomic effects on child health outcomes. These include gender, number of older siblings, a household wealth index, mother’s height, mother’s education, child ethnicity, level of antenatal care, and weeks premature at birth.⁸ In a robustness analysis, I also make use of other YLS data in attempt to evaluate alternate mechanisms that may be driving results. First, I use individual level data on the household’s main source of drinking water. Reported responses were grouped into four sources—unprotected (e.g. river, pond, unprotected well), piped directly into private dwelling/yard, public standpipe/tubewell, or other source. Second, I use community survey data to construct a measure of prenatal exposure to seasonal increases in local labor demand. Specifically, I use responses to the following survey question:

In which months of the year is there relatively more work to do?

With this data, I construct the new exposure measure in an analogous fashion as my measure

⁷Prevalence of child stunting is the percentage of children whose height-for-age is more than two standard deviations below the median for the international reference population. Wasting is defined analogously using weight-for-height.

⁸YLS provides a constructed household wealth index for each of the three survey rounds. After taking the log and standardizing each to have mean zero and variance one, I use the standardized average across non-missing values as my household wealth index. Mother’s education reported as “adult literacy” included in the one to four years of education category. Level of antenatal care was derived by YLS based on time of first visit, number of visits, and whether mothers had a tetanus injection.

of prenatal exposure to seasonal food scarcity. Descriptive statistics for household and child characteristics are presented in Panel B of Table 3.1.

3.3. EMPIRICAL STRATEGY

Empirical analysis of the effects of prenatal exposure to seasonal food scarcity on child health outcomes is based on the following benchmark specification:

$$Y_{idc} = \alpha + \delta Exp_{dc} + \beta X_{idc} + \theta_c + \mu_m + u_{idc}, \quad (3.1)$$

where Y_{idc} is a health outcome for individual i , born on date d , in community c ; Exp_{dc} is my measure of prenatal exposure to seasonal food scarcity; X_{idc} are household and child characteristics; θ_c is a fixed effect for community of residence; μ_m is a fixed effect for month of birth; and u_{idc} is a random error term. I estimate the equation separately for child height and body mass outcomes measured at approximately one, five, and eight years of age. The coefficient of interest is that on prenatal exposure to relative food scarcity δ . Note that the exposure measure varies by date of birth d and community of residence c . Throughout the analysis, exposure days are expressed in thirty day units so coefficients can be interpreted as approximate monthly effects. Household and child characteristics include age of the child in months when the outcome was measured, a household wealth index, number of older siblings and dummies for gender, mother's education, mother's height, child ethnicity, level of antenatal care, and weeks premature at birth.⁹

Identification relies on the assumption that prenatal exposure to reported months of

⁹Approximately 11.8% of individuals were missing data on mother's height and 6.5% on level of antenatal care. I avoid dropping these observations in the reported analysis by including dummy indicators for those missing either measure. For maternal height, I implement this by including dummy variables for each decile of the distribution opposed to using a continuous measure. Results are also robust to the exclusion of these two controls.

Table 3.1: Descriptive Statistics: Health Outcomes and Other Data

| | Mean | S.D. | Obs. | | Mean | S.D. | Obs. |
|--|-------|------|------|---|-------|------|------|
| <i>Panel A — Child Health Outcomes</i> | | | | | | | |
| Height (cm) | | | | Body Mass Index (BMI) | | | |
| Age 1 | 70.8 | 5.48 | 1849 | Age 1 | 0.0 | 1.00 | 1756 |
| Age 5 | 103.7 | 5.38 | 1810 | Age 5 | 0.0 | 1.00 | 1810 |
| Age 8 | 120.4 | 6.58 | 1784 | Age 8 | 0.0 | 1.00 | 1784 |
| <i>Panel B — Household and Child Characteristics</i> | | | | | | | |
| Male | 0.53 | 0.50 | 1899 | Antenatal Care | | | |
| Older Siblings | 2.64 | 2.38 | 1899 | None | 0.56 | 0.50 | 1775 |
| Wealth Index | 0.00 | 1.00 | 1899 | Low | 0.17 | 0.37 | 1775 |
| Mother's Height (cm) | 158.6 | 5.88 | 1675 | Medium | 0.11 | 0.32 | 1775 |
| Mother's Education | | | | High | 0.16 | 0.36 | 1775 |
| None | 0.53 | 0.50 | 1890 | Weeks Premature | | | |
| 1 to 4 years | 0.22 | 0.41 | 1890 | None | 0.92 | 0.24 | 1899 |
| 4 to 8 years | 0.15 | 0.36 | 1890 | 1 to 2 weeks | 0.04 | 0.20 | 1899 |
| >8 years | 0.09 | 0.29 | 1890 | >2 weeks | 0.02 | 0.14 | 1899 |
| Child Ethnicity | | | | Unknown | 0.02 | 0.15 | 1899 |
| Amhara | 0.29 | 0.45 | 1899 | Water Source | | | |
| Gurage | 0.08 | 0.27 | 1899 | Unprotected | 0.44 | 0.50 | 1899 |
| Oromo | 0.21 | 0.41 | 1899 | Private Pipe | 0.11 | 0.31 | 1899 |
| Tigrian | 0.23 | 0.42 | 1899 | Public Standpipe/Well | 0.40 | 0.49 | 1899 |
| Wolavta | 0.05 | 0.21 | 1899 | Other | 0.05 | 0.21 | 1899 |
| Other | 0.14 | 0.35 | 1899 | Prenatal Days Exposed to High Labor Demand | 121.0 | 67.8 | 1899 |

Source: Young Lives Study, Ethiopia, young cohort. Sample of children without missing community data on seasonal food scarcity (n = 1,899).

food scarcity is uncorrelated with any unobserved determinants of examined child health outcomes. It is clearly the case that community of residence is likely to be correlated with both health outcomes and the exposure measure, as climatic conditions and household demographics vary considerably across Ethiopia. However, the inclusion of community dummy variables ensures that effects associated with geographic area are controlled for. An additional concern is the existence of unobserved socioeconomic or demographic determinants of child health that correlate with seasonal patterns of fertility, and hence the exposure measure. However, the inclusion of month of birth dummies controls for seasonal effects that occur at the country level and are not related to exposure to food scarcity.¹⁰ It is still conceivable that seasonal fertility patterns could correlate with unobserved characteristics within communities, but as detailed in later robustness analysis, I find no evidence of this based on the rich set of observable household characteristics available.

Even under the identifying assumption, interpretation of the coefficient of interest δ requires careful consideration. Due to collinearity, δ is a measure of the effect of exposure during the approximately nine months prior to birth relative to the first three months after birth (i.e. the first three months after birth is the reference period). However, imposing a relatively mild assumption can allow for further interpretation of the empirical estimates. Specifically, I assume that the effects of exposure are weakly negative regardless of whether exposure occurs during pregnancy or the months just after birth. Under this assumption, I interpret the empirical δ estimates as the minimum total effect of prenatal exposure to food scarcity according to the following proposition:

Proposition. *If the effects of exposure to relative food scarcity the year following conception are weakly negative during pregnancy and after birth, then δ is the minimum effect (in absolute terms) of prenatal exposure to food scarcity. Proof: see Appendix C.*

In addition to the benchmark specification, I examine heterogeneity of effects across a

¹⁰Month of birth dummies also help control for nonlinear growth in children that may not be captured by the continuous age of child control.

number of dimensions to shed further light on the relevant mechanisms at work. I analyze how effects vary over household wealth, maternal education, and the timing of exposure over stages of gestation. Results are compared with previous findings in the medical and economic literatures. I also examine heterogeneity across sources of water supply and exposure to seasonal variation in work availability to assess if results may be partially operating through access to clean water or changes in maternal labor supply in congruence with food availability. Lastly, I discuss and evaluate the potential influence of selective mortality on the basis of prenatal exposure to seasonal food scarcity. This may be a particular concern because of the high infant mortality rates in Ethiopia.

3.4. RESULTS

3.4.1 Benchmark Results

The main results from the benchmark specification are presented in Table 3.2. Under the assumptions specified in the previous section, I interpret reported coefficients as lower (absolute) bounds on the total effects of exposure to seasonal food scarcity on corresponding health outcomes. The first three columns report the estimated exposure effect on child height measured at approximately age one, five, and eight. The last three columns give analogous results for the standardized measure of body mass.

Column (1) shows a lower bound on the effect of exposure on age one height is not identified at a statistically significant level. However, by age five, the estimated coefficient has greatly increased in magnitude and is statistically significant. The coefficient implies, holding other independent variables constant, an additional month of prenatal exposure to food scarcity leads to a decrease in height of at least 0.31 cm. Moreover, column (3) shows the magnitude of the estimated effect increases to 0.41 cm by age eight. This pattern is

consistent with a divergence of heights between ages one and eight on the basis of prenatal exposure to food scarcity. This would imply that parents are unable and/or unwilling to fully make up for the early effects of exposure. This is consistent with the view of gestation as a “sensitive” period of child development in which nutritional and other inputs are difficult to substitute for in later stages of life (Cunha and Heckman 2007).

Table 3.2: Effects of Prenatal Exposure to Seasonal Food Scarcity on Child Health Outcomes

| | $Height_1$ | $Height_5$ | $Height_8$ | BMI_1 | BMI_5 | BMI_8 |
|-------|------------|------------|------------|---------|---------|---------|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Exp | -0.051 | -0.307* | -0.407*** | -0.034 | -0.010 | 0.010 |
| | (0.210) | (0.167) | (0.118) | (0.027) | (0.028) | (0.020) |
| Obs | 1,840 | 1,804 | 1,776 | 1,748 | 1,804 | 1,776 |
| R^2 | 0.403 | 0.270 | 0.221 | 0.294 | 0.159 | 0.113 |

Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: height and body mass at age one, five, and eight. Reported independent variable: prenatal exposure to seasonal food scarcity (Exp). Additional independent variables in all regressions: age of child in months, wealth index, number of older siblings, and dummies for gender, mother’s height, mother’s education, ethnicity, antenatal care, weeks premature, month of birth, and community.

The last three columns in Table 3.2 show that non-zero lower bounds on the effects of exposure on body mass index are not identified at any age. As body mass is essentially a measure of weight-for-height, these estimates are consistent with height and weight being measures of the variation of health inputs in the long-run and short-run, respectively. Specifically, prenatal exposure has a long-term impact on health as demonstrated by the effects on childhood height but, after controlling for height, has no significant impact on the short-term health measure (weight). Furthermore, the life-cycle impact of early malnourishment on body mass is not fully understood. In general, studies have found that poor early nutrition can lead to obesity later in life (Black et al. 2013). Such a mechanism is also consistent with the weak effects on body mass as compared to height found in this chapter. However, I will return to this discussion as further insights are revealed when examining how the timing of exposure during the gestational period effects health outcomes at alternate ages.

3.4.2 Socioeconomic Heterogeneity

In order to gain further insight on the mechanism at work in my baseline specification, I next examine how the effects of prenatal exposure to seasonal food scarcity vary with socioeconomic status. Specifically, I estimate heterogeneity in effects across household wealth and level of maternal education. As the benchmark specification identified no significant relationships between overall exposure and body mass, I limit my analysis here to child height outcomes.

Panel A of Table 3.3 reports results when adding an interaction between the exposure measure and the household wealth index to the benchmark specification. Children in wealthier households were significantly less adversely affected by prenatal exposure to reported food scarcity. Moreover, the heterogeneity is quantitatively substantial. For example, the estimated coefficients imply the minimum decrease in height by age five from a month of exposure is 0.62 cm for a child from the tenth percentile of the wealth distribution compared to 0.11 cm from the ninetieth percentile. By age eight, the estimated magnitude of the effects has increased to 0.76 cm and 0.17 cm, respectively. These findings are consistent with the related empirical literature which generally find stronger long term effects of early health shocks on poor households (e.g. Currie and Hyson 1999). In the context of this study, results suggests that poor families may be particularly vulnerable to variation in food availability in Ethiopia.

There are several possible channels through which household wealth may influence the effects of exposure. First, wealthier families may be better equipped to smooth consumption during pregnancy over fluctuations in food prices or availability. As a result, children from these households may directly experience a smaller exposure shock in-utero. Alternatively, wealthier parents may have the financial means to make additional remedial investments after birth. In this instance, even if actual in-utero exposure is similar across wealth levels, exposed children from wealthier families may “catch up” to unexposed children through

Table 3.3: Heterogeneity of Effects by Wealth and Maternal Education

| | <i>Height</i> ₁ | <i>Height</i> ₅ | <i>Height</i> ₈ |
|--|----------------------------|----------------------------|----------------------------|
| | (1) | (2) | (3) |
| <i>Panel A — Heterogeneity by Household Wealth</i> | | | |
| <i>Exp</i> | -0.064 (0.226) | -0.337* (0.187) | -0.439*** (0.139) |
| <i>Exp</i> × <i>Wealth</i> | 0.101 (0.084) | 0.202*** (0.069) | 0.233* (0.117) |
| <i>Panel B — Heterogeneity by Mother's Education</i> | | | |
| <i>Exp</i> × <i>MomEd</i> | | | |
| None | -0.080 (0.217) | -0.430** (0.202) | -0.594*** (0.171) |
| 1 to 4 years | -0.085 (0.242) | -0.314 (0.220) | -0.419* (0.212) |
| 5 to 8 years | 0.076 (0.261) | -0.224 (0.248) | -0.078 (0.247) |
| >8 years | -0.062 (0.302) | 0.035 (0.345) | -0.146 (0.401) |
| <i>Obs</i> | 1,840 | 1,804 | 1,776 |

Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: height at age one, five, and eight. Reported independent variables: interaction between prenatal exposure to seasonal food scarcity and wealth index (Panel A) or mother's education (Panel B). Additional independent variables in all regressions: age of child in months, wealth index, number of older siblings, and dummies for gender, mother's height, mother's education, ethnicity, antenatal care, weeks premature, month of birth, and community.

remedial health investments by the time the outcomes are measured. In either of these two cases, it is important to note that the reported results may underestimate the total costs of exposure as there may be additional utility costs due the reallocation of resources in response to the shock. However, in contrast to the above channels which focus on responsive investments by parents, the relationship between wealth and exposure could partially be a mechanical feature of child health “production.” For example, many empirical and theoretical studies argue that there are diminishing marginal returns to health investments. Thus, all else equal, children with higher baseline levels of investment would be less adversely affected

by an equivalent decrease in health inputs. In the context of this study, it seems plausible that wealthier children could have higher baseline levels of prenatal health inputs compared to their less wealthy counterparts (e.g. wealthier mothers have more substantial or varied diets). If so, even if all mothers respond to exposure with equivalent level declines in health inputs, wealthier children could be less adversely affected by exposure.

I next examine the heterogeneity of effects by level of maternal education based on four schooling categories—none, lower primary (1-4 years), upper primary (5-8 years), and secondary plus (>8 years). For ease of interpreting results, I drop the exposure measure (*Exp*) from the benchmark specification and add interaction terms between exposure and indicators for each level of maternal education. In this way, coefficients can be directly interpreted as estimated effects of exposure for each category. Results are reported in Panel B of Table 3.3. The estimated minimum effect of an additional month of exposure on a child born to a mother with no education is -0.43 cm by age five and -0.59 cm by age eight. The only other statistically significant effect is on height at age eight for children born to mothers with one to four years of education (-0.42 cm). Statistically significant lower bounds at any age can not be identified for children born to more educated mothers. However, although not precisely estimated, there is a clear pattern of diminishing effects moving up the full set of maternal education indicators for height at age five and eight. To the extent that more highly educated mothers come from wealthier families, similar channels as detailed above could be partially explaining these results. However, it could also be the case that maternal cognitive capacity directly influences the effects of exposure. For example, more highly educated mothers may have partially shielded their fetus from the effects of external food scarcity. This would be consistent with much of the empirical evidence that maternal education strongly impacts child health outcomes through improved child-care practices and attitudes towards reproductive behavior (e.g. Thomas et al. 1991; Glewwe 1999).

3.4.3 Timing of Prenatal Exposure

In addition to the benchmark measure of total prenatal exposure, it is also insightful to analyze how effects are influenced by the timing of exposure over gestational periods of development. In analyzing these differences, I follow the common practice of delineating the prenatal period into three trimesters of pregnancy. These trimesters roughly coincide with embryogenesis (first trimester), fetal development (second trimester), and a perinatal period (third trimester). The empirical medical and economic literature examining prenatal shocks during alternate gestational periods is substantial and quite varied. However, there is considerable evidence that long-term health outcomes such as diabetes and heart disease may be particularly sensitive to insults during the first trimester of gestation.¹¹ In regards to short-term effects, studies have shown significant impact on birthweight from shocks during all stages of prenatal development, though a majority are focused around mid to late term shocks (Currie and Almond 2011).

In order to empirically examine the importance of the timing of prenatal exposure to seasonal food scarcity in Ethiopia, I translate my baseline exposure measure into distinct trimester measures. In practice, I drop the total exposure measure (*Exp*) from the benchmark specification and add three measures indicating the number of exposure days during each trimester. Results from this specification are reported in Table 3.4. The first three columns show the effects of exposure by trimester of gestation on child height at ages one, five, and eight. For example, exposure to an additional month of reported food scarcity during the first trimester reduces child height by an estimated 0.44 cm by age five. While coefficients are negative for all trimesters at age five, the magnitude of the estimate is considerably larger and statistically significant only for the first trimester. By age eight, effects are significant from exposure during all stages of gestation, but remain strongest for the first trimester. As age five and eight measures were taken several years after gestation, I view this as consistent with the empirical findings that health insults during the first trimester

¹¹See, for example, the summary of studies in Almond and Mazumder (2011), Table A1.

have stronger effects on long-term health outcomes.

Table 3.4: Effects of Exposure by Trimester of Gestation

| | $Height_1$ | $Height_5$ | $Height_8$ | BMI_1 | BMI_5 | BMI_8 |
|---------------|-------------------|---------------------|---------------------|--------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| 1st Trimester | -0.199 (0.173) | -0.435** (0.169) | -0.626** (0.267) | 0.021 (0.040) | 0.038 (0.037) | 0.059 (0.056) |
| 2nd Trimester | -0.025 (0.245) | -0.296 (0.198) | -0.353** (0.128) | -0.050* (0.026) | -0.028 (0.030) | -0.008 (0.016) |
| 3rd Trimester | -0.042 (0.152) | -0.240 (0.195) | -0.449* (0.220) | -0.008 (0.038) | 0.023 (0.041) | 0.045 (0.034) |
| <i>Obs</i> | 1,840 | 1,804 | 1,776 | 1,748 | 1,804 | 1,776 |
| R^2 | 0.404 | 0.271 | 0.222 | 0.296 | 0.160 | 0.114 |

Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: height and body mass at age one, five, and eight. Reported independent variables: binary indicators for "majority" exposure to seasonal food scarcity by trimester. Additional independent variables in all regressions: age of child in months, wealth index, number of older siblings, and dummies for gender, mother's height, mother's education, ethnicity, antenatal care, weeks premature, month of birth, and community.

The last three columns of Table 3.4 give the estimated trimester effects on child body mass. At age one, estimates on the last two trimesters are negative, though the coefficient is only statistically significant for the second trimester. This implies that an additional month of exposure to food scarcity during the second trimester reduces child body mass at age one by an estimated 0.05 standard deviations. As age one measurements are taken relatively soon after birth, I view these results as consistent with the empirical evidence that mid to late term shocks have a stronger impact on birthweight. However, unlike effects on child height, this result does not intensify with age, as the magnitude and statistical significance fades away by age five. This is again consistent with body mass being a measure of health inputs in the relatively short-term.

3.5. ASSESSING ALTERNATE CHANNELS

3.5.1 Water Accessibility and Maternal Labor Supply

Due to the explicit nature of the survey data used to construct my exposure measure, I argue that my results are primarily operating through nutritional intake fluctuations due to seasonal variations in food availability and/or prices. However, it is conceivable that my results could stem from other channels as well. For example, if seasonal variation in food scarcity is highly correlated with water supply quality for some households, results could be driven by the adverse disease environment that is known to accompany limited access to clean water. In order to evaluate the evidence on the commonly proposed confounding channels in developing countries—water quality and maternal labor supply—I examine heterogeneity of effects by household sources of drinking water and prenatal exposure to increased labor demand.

I begin by re-estimating the benchmark specification with the inclusion of an indicator for household source of drinking water as well as its interaction with prenatal exposure to reported food scarcity. I also include an interaction between the exposure measure and the wealth index to help ensure that water source interactions are not simply reflecting exposure effect heterogeneity by household wealth. Panel A of Table 3.5 reports results for the altered specification. The water source reference group are those families who access water through unprotected sources (e.g. river, pond, unprotected well). If access to clean drinking water was indeed a confounding channel, we would expect households with better access to protected water sources to be less adversely affected by the exposure measure (i.e. positive interaction effects). Results indicate no statistically significant difference in the effects of exposure between the reference group and those who access water through private pipes, public standpipes/wells, or other sources. I will note, however, that in comparison to the main exposure effect, the magnitude of several interaction coefficients are positive

and substantial for the private pipe and other sources categories. However, the coefficients are imprecisely estimated as these groups comprise only about 11% and 5% of the sample, respectively. Moreover, as these categories are highly correlated with wealth, interaction coefficients could also be reflecting socioeconomic heterogeneity not fully captured by the wealth index interaction. A majority of sample households that did not obtain water through unprotected sources did so through public standpipes or wells. For this group, interaction estimates are both statistically insignificant and small in comparative magnitude. Thus, at least tentatively, I find little evidence suggesting that results are being substantially driven by seasonal variations in water supply quality.

An alternative concern is that agriculture production cycles may be correlated with maternal labor supply. Researchers have argued that seasonal changes in maternal labor supply during pregnancy could impact fetal development (e.g. Strand et al. 2011). This may be especially true in developing countries where labor is often concentrated in the physically demanding agricultural sector. Although I do not have a direct measure of maternal labor decisions throughout pregnancy, I use my measure of prenatal exposure to seasonal increases in local labor demand (*Labor*) as a proxy for increases in maternal labor supply. As shown in Panel B of Table 3.5, adding this proxy to the benchmark specification has little impact on estimated effects of exposure to seasonal food scarcity. If anything, the magnitude of impacts increase slightly suggesting benchmark results may be biased towards zero due to correlations with maternal labor supply. This is perhaps unsurprising as it is quite plausible that maternal labor supply is negatively correlated with food scarcity as well as child health outcomes. It may be of interest to note that all point estimates on the maternal labor supply proxy are negative and increasing in magnitude over time. As such, results could still be consistent with the conjecture of maternal labor decisions as a separate channel through which prenatal environment affects long-term health trajectories in Ethiopia.

Table 3.5: Heterogeneity by Water Source and Exposure to Seasonal Labor Demand

| | $Height_1$ | $Height_5$ | $Height_8$ |
|---|-------------------|--------------------|----------------------|
| | (1) | (2) | (3) |
| <i>Panel A — Heterogeneity by Source of Water</i> | | | |
| <i>Exp</i> | 0.040 (0.251) | -0.387* (0.221) | -0.491** (0.215) |
| <i>Exp</i> \times <i>Wealth</i> | 0.147 (0.097) | 0.167* (0.084) | 0.197 (0.140) |
| <i>Exp</i> \times <i>WaterSource</i> | | | |
| Private Pipe | -0.213 (0.187) | 0.236 (0.300) | 0.219 (0.340) |
| Public Standpipe/Well | -0.176 (0.135) | 0.054 (0.207) | -0.005 (0.219) |
| Other | -0.081 (0.396) | -0.048 (0.507) | 0.357 (0.355) |
| <i>Panel B — Effects of Exposure to Seasonal Labor Demand</i> | | | |
| <i>Exp</i> | -0.060 (0.205) | -0.324* (0.170) | -0.445*** (0.131) |
| <i>Labor</i> | -0.035 (0.118) | -0.069 (0.147) | -0.158 (0.133) |
| <i>Obs</i> | 1,840 | 1,804 | 1,776 |

Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: height at age one, five, and eight. Reported independent variables: prenatal exposure to seasonal food scarcity (both panels), interaction with wealth and water source (Panel A), and exposure to increased seasonal labor demand (Panel B). Additional independent variables: age of child in months, wealth index, number of older siblings, and dummies for water source (Panel A only), gender, mother's height, mother's education, ethnicity, antenatal care, weeks premature, month of birth, and community.

3.5.2 Seasonal Patterns of Fertility

A remaining concern is the possibility that seasonal patterns of fertility within communities could be biasing the results of the empirical analysis. Recall that while inclusion of month of birth dummies controls for seasonal effects that occur at the country level, it is still conceivable that seasonal fertility patterns could correlate with unobserved characteristics within communities. If, for example, the pregnancies of wealthier women correlate with periods when food is relatively plentiful, then results could be attributed to differences in resources available to the child as opposed to exposure to food scarcity. Moreover, studies have documented seasonal patterns of fertility across a variety of countries (e.g. Rajagopalan et al. 1981; Panter-Brick 1996; Artadi 2005; Buckles and Hungerman 2013). In the developing world, these patterns have been most commonly linked to the influence of agricultural cycles on female labor supply, seasonality of marriage, and male migration.

In order to evaluate seasonal fertility patterns across the study sample, I begin by examining the timing of births by calendar date (see Figure 3.5). There is a small decline in births in August-September 2001 and January-March 2002, both followed by a period of somewhat higher birthrates. However, there is no discernible correlation between these birthrate patterns and the aggregated community food scarcity data shown in Figure 3.2. Nonetheless, to more rigorously evaluate the influence of fertility patterns associated with family demographics, I estimate the relationship between my measure of exposure to food scarcity and household characteristics. Specifically, I estimate the following equation:

$$ExpDays_{idc} = \alpha + \beta X_{idc} + \theta_c + u_{idc} \quad (3.2)$$

where X are the same set of child and household characteristics used in the baseline specification and θ are community of residence indicators.

Panel A of Table 3.6 reports the estimated coefficients on observed characteristics from equation 3.2. Virtually none of the child or household characteristics are related with the

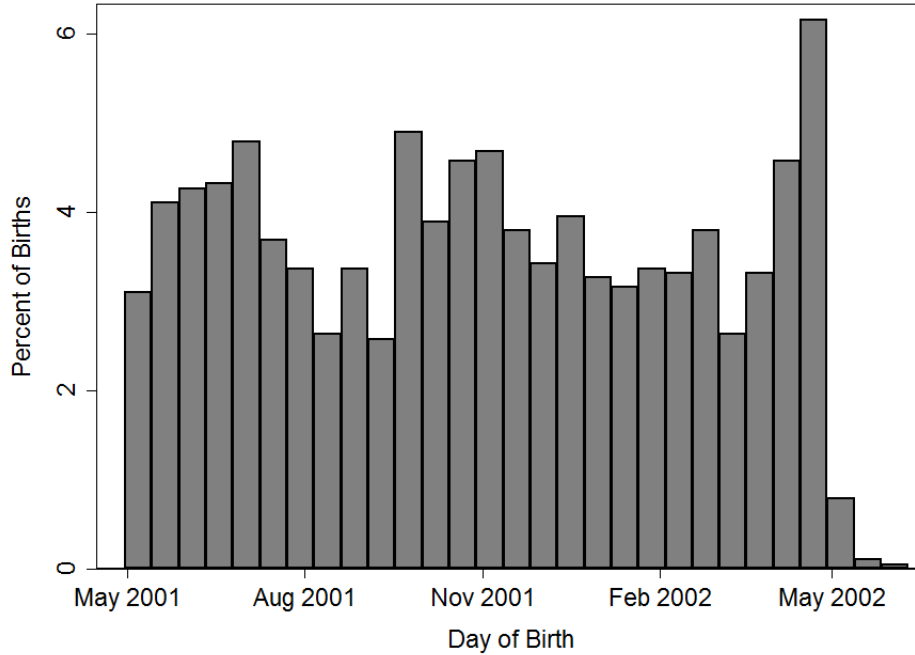


Figure 3.5: Date of Birth. Source: Author’s calculations using data from Young Lives Study, Ethiopia. Histogram with 2 week window for each bin.

number of exposure days at conventional significance levels. The only exception is a negative correlation with premature birth. However, this is be expected given the gestation period for preterm births is by definition shorter than full-term births. An F -test cannot reject the null-hypothesis that reported coefficients, other than those on premature birth, are equal to zero. Moreover, according to a likelihood ratio test, inclusion of the other household characteristics does not significantly improve the fit of the specification. As such, based on observed characteristics, there is no evidence of substantial selection based on demographic or socioeconomic seasonal patterns of fertility.

An alternate source of seasonal fertility bias could emerge as a result of unplanned pregnancies. Suppose, for example, that parents attempt to plan pregnancies around seasonal variations in food availability because they *believe* exposure is “bad” for their unborn child. In this case, children exposed to heavy amounts of food scarcity are more likely to be the result of unplanned pregnancies. Moreover, several studies have linked unplanned pregnancies to negative child outcomes including health status (e.g. Kost et al. 1998; Do and Phung

Table 3.6: Dependent Variable: Prenatal Days of Exposure to Seasonal Food Scarcity

| <i>Panel A — Coefficients on Observed Characteristics</i> | | | | | |
|---|-----------|---------|---------------------------|--------|------------------|
| Male | -1.804 | (1.578) | Antenatal Care | | |
| Older Siblings | -0.377 | (0.351) | Low | 1.371 | (2.319) |
| Wealth Index | -0.794 | (1.276) | Medium | 1.569 | (2.786) |
| Mother's Education | | | High | -2.453 | (2.758) |
| 1 to 4 years | 2.717 | (2.108) | Missing | 1.459 | (3.447) |
| 4 to 8 years | 3.234 | (2.623) | Mother's Height (decile) | | |
| >8 years | 5.431 | (3.413) | 2 | 2.302 | (3.553) |
| Weeks Premature | | | 3 | 4.171 | (3.905) |
| 1 to 2 weeks | -1.288 | (3.940) | 4 | -2.001 | (3.669) |
| >2 weeks | -15.46*** | (5.539) | 5 | 1.153 | (3.679) |
| Unknown | -17.12*** | (5.541) | 6 | 1.370 | (3.545) |
| Child Ethnicity | | | 7 | 1.464 | (3.874) |
| Amhara | -7.063 | (4.990) | 8 | -2.841 | (3.696) |
| Gurage | -5.517 | (6.488) | 9 | 0.090 | (3.654) |
| Oromo | -5.873 | (4.665) | 10 | 0.199 | (3.755) |
| Tigrian | -9.290 | (6.868) | Missing | -1.380 | (3.485) |
| Wolavta | 8.568 | (10.48) | | | |
| <i>Obs</i> | 1890 | | <i>F</i> (25, 1840) | 0.76 | <i>p</i> < 0.800 |
| <i>R</i> ² | 0.540 | | <i>LR</i> : χ^2 (25) | 19.34 | <i>p</i> < 0.780 |
| <i>Panel B — Coefficients on Unplanned Pregnancy</i> | | | | | |
| Wanted Pregnancy | | | | | |
| No | 2.370 | (1.737) | <i>Obs</i> | 1890 | |
| Missing | -0.750 | (4.612) | <i>R</i> ² | 0.540 | |

Standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. All regressions include community fixed-effects. Reported *F*-test for joint significance of all independent variables in Panel A except weeks premature. Reported likelihood ratio test for null model including community fixed-effects and weeks premature. Additional independent variables in Panel B: age of child in months, wealth index, number of older siblings, and dummies for gender, mother's height, mother's education, ethnicity, antenatal care, weeks premature, month of birth, and community.

2010; Lokshin and Radyakin 2012). Under this scenario, the correlation between exposure and child health could be the result of a higher proportion of unplanned pregnancies during times of relative food scarcity.

In relation to unplanned pregnancies, the YLS survey asked participants the following question:

At the time you became pregnant with ‘NAME’ did you want to become pregnant?

Based on the replies, about 35% of pregnancies were “unwanted” with another 5% of responses missing due to the mother not being present for the interview. I use these data to evaluate the extent to which undesired pregnancies could be biasing my empirical results. Specifically, I re-estimate equation 3.2 but also include an indicator variable that takes a value of one if the pregnancy was reportedly “unwanted”. I also include a variable to indicate if the response to pregnancy desirability was missing. As shown in Panel B of Table 3.6, the relationship between undesired pregnancies and days of exposure to seasonal food scarcity is not statistically significant. The point estimate is also quantitative small—an undesired pregnancy is correlated with an increase in exposure of about two days. As such, I find little evidence of selection based on seasonal concentrations of unplanned pregnancies.

3.5.3 Mortality Selection

A final concern is the possibility of bias due to selective mortality on the basis of prenatal exposure to seasonal food scarcity. The basic problem is that a given outcome is only observed for children that survive to the age of measurement. If prenatal exposure has differentiated mortality effects on children, the composition of survivors may be different than it would have been in absence of exposure. However, mortality selection from a negative in-utero health shock will generally result in estimates that understate the magnitude of effects (Almond and Currie 2011). For example, if only the healthier or more robust of the exposed children survive, then selective mortality of unhealthy children would be biasing

results towards zero. Nonetheless, while I cannot directly evaluate the magnitude of selective mortality that occurred in-utero, I can examine the evidence of selection bias between rounds of data collection.

Approximately 4.7% of children were missing health outcomes at age five, and 6.1% at age eight. Unfortunately, I cannot identify the proportion of missing data that occurs as an explicit result of child mortality. However, the estimated child mortality rate between ages one and five for Ethiopia in 2006 was 3.7%, suggesting a potentially significant role for mortality in generating missing survey data (World Bank 2015). Moreover, while receiving less attention in the literature, other proximate causes of missing data could bias results through similar mechanisms as mortality selection.

In order to evaluate the potential role of this type of selection bias on results, I estimate the probability of having missing data at ages five or eight using a simple linear probability model:

$$Miss_{idca} = \alpha + \delta Exp_{dc} + \pi H_{idc} + \kappa (Exp_{dc} \times H_{idc}) + \beta X_{idc} + \mu_m + \theta_c + u_{idc},$$

where $Miss_{idca}$ is an indicator for a missing health outcome at age a , H_{idc} is a measure of child health during the first round of data collection, and other explanatory variables are as in the benchmark specification.¹² In practice, I use height and body mass at age one as alternate proxies for child health during the first round. The coefficient of interest is that on the interaction between prenatal exposure and age one health. A significant coefficient would reject the null hypothesis that exposure has no differentiated effects on the probability of missing outcomes based on early health status.

Table 3.7 shows the relevant results from the probability estimates using first round height or body mass as early measures of child health. All interaction coefficients are statistically insignificant at conventional levels, providing no evidence of substantial selection bias due to

¹²Significance of results are robust to the use of a logistic regression. I present the linear probability model for simplicity in interpreting interaction terms.

mortality or other forces resulting in missing health outcomes. Moreover, although imprecisely estimated, point estimates for all interaction coefficients are negative. This tentatively suggests that, if anything, healthier children are more likely to survive exposure than their less healthy counterparts. This would be consistent with the results from the benchmark specification underestimating the effects of prenatal exposure to seasonal food scarcity.

Table 3.7: Heterogeneity of Effects of Exposure on Probability of Missing Outcomes

| | <i>Miss</i> ₅ | <i>Miss</i> ₅ | <i>Miss</i> ₈ | <i>Miss</i> ₈ |
|---|--------------------------|--------------------------|--------------------------|--------------------------|
| | (1) | (2) | (3) | (4) |
| <i>Exp</i> × <i>Height</i> ₁ | -0.044 (0.057) | | -0.038 (0.059) | |
| <i>Exp</i> × <i>BMI</i> ₁ | | -0.005 (0.005) | | -0.001 (0.005) |
| <i>Obs</i> | 1,840 | 1,748 | 1,840 | 1,748 |
| <i>R</i> ² | 0.043 | 0.040 | 0.047 | 0.045 |

Coefficients of linear probability model reported. Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: missing outcomes at age one and five. Reported independent variables: interaction between prenatal exposure to seasonal food scarcity and round one height (meters) or body mass. Additional independent variables in all regressions: height (round one), body mass (round one), age of child in months (round one), wealth index, number of older siblings, and dummies for gender, mother’s education, ethnicity, antenatal care, weeks premature, month of birth, and community.

3.6. CONCLUSIONS

This chapter presents novel evidence on the impact of prenatal nutritional environment on later childhood health outcomes. Among a cohort of Ethiopian children, I find that prenatal exposure to months of reported seasonal food scarcity had a significant negative effect on height by age five. Furthermore, these effects strengthen by age eight and are stronger when exposure is concentrated in the first trimester, supporting early gestation as a “sensitive” period of child development. In contrast, effects on child body mass are strongest when

exposure is concentrated in the second trimester and tend to fade with time. Consistent with other empirical studies, effects are also stronger for poorer children and those born to less educated mothers. I also find no evidence that results are driven by commonly proposed seasonal factors other than prenatal nutritional environment.

The impact of seasonal food scarcity on prenatal development has important policy implications in Ethiopia and throughout much of the developing world. In the long-run, addressing the source of seasonal food insecurity likely involves substantial public investment in transport infrastructure, storage and processing technologies, promotion of alternate crop varieties, and agricultural market organization (World Bank 2012). In the meantime, social safety net programs can serve to combat the impact of seasonal food scarcities in the short-run. However, these programs come at the cost of diverting limited public funds from long-term investments and potentially creating a chronic dependency on food aid. As such, understanding which populations are particularly vulnerable to seasonal food insecurity can help efficiently target relief interventions.

In Ethiopia, policies such as the Productive Safety Net Programme (PSNP), launched in 2005, already aim to provide predictable support for seasonal variations in food availability. Further targeting benefits towards pregnant women or those of child rearing age could be a low-cost but effective modification to such a program. Information or family planning campaigns could also be modified to emphasize the impact seasonal variations in maternal diets can have on fetal development. Other low-cost interventions such as distribution of nutrient rich season-specific recipes or improved home-based preservation technologies are being piloted in other developing countries (Wijesinha-Bettoni et al. 2013). My findings suggest such policies that mitigate the effects of seasonal food scarcities on the prenatal nutritional environment could have significant and long lasting impacts on child health.

APPENDIX A

DEFINITION OF STATIONARY COMPETITIVE EQUILIBRIUM

For ease of notation let $\zeta = \{b, i, \theta\}$. Given tax rate τ and relative prices P_i and P_m , a stationary competitive equilibrium is a set of household decision rules:

$$\{g_c(\zeta), g_s(\zeta), g_q(\zeta), g_{i'}(\zeta), g_{a'}(\zeta), g_g(\zeta), g_h(\zeta)\},$$

a household value function $V(\zeta)$, factor prices (w, r) , government subsidies (p_i, p_e) , and a time invariant distribution of households across states Ψ , such that:

1. Given prices, government subsidies, and tax rates, the value and policy functions solve the household decision problem.
2. Given prices, firms maximize profits

$$(a) \quad w = (1 - \alpha) AK^\alpha H^{-\alpha}$$

$$(b) \quad r = \alpha AK^{\alpha-1} H^{1-\alpha}$$

3. Markets Clear

$$(a) \quad K = \int g_{a'}(\zeta) d\Psi(\zeta)$$

$$(b) \quad H = \int [(\psi(1 - g_s(\zeta)) + 1 + \kappa) f(i', g_s(\zeta), g_q(\zeta), \theta)] d\Psi(\zeta) - \int g_{\bar{h}}(\zeta) d\Psi(\zeta)$$

$$(c) \quad Y = \int [g_c(\zeta) + g_{a'}(\zeta) + P_g g_g(\zeta) + P_i g_{i'}(\zeta)] d\Psi(\zeta) - (1 - \delta) K = AK^\alpha H^{1-\alpha}$$

4. Government budget is balanced

$$(a) \quad \tau(wH + rK) = \int [p_q g_q(\zeta) + p_i g_{i'}(\zeta)] d\Psi(\zeta)$$

APPENDIX B

ALGORITHM TO COMPUTE STATIONARY EQUILIBRIUM

1. Make initial guesses for the wage rate w , interest rate R , and tax rate τ .¹³
2. Compute household decision rules (see below).
3. Simulate a dynasty for 50,000 generations and discard the first 5,000 as a burn in period. Aggregate assets, human capital, and education and early health expenditures across the remaining generations.
4. Use the solutions to firm's problem and government budget constraint to update w , R , and τ . Iterate from step 2 until convergence.

Computing household decision rules All household decision rules are computed for a discrete exogenous grid of (b, i, θ) . For computation, I use a modified version of the endogenous gridpoint method with occasionally binding constraints proposed by Hintermaier and Koeniger (2010). The basic strategy is to combine the state vectors for early health i and ability θ with an exogenous grid of early health choices i' and then back out the remaining implied decision rules and an endogenous state wealth grid \hat{b} . These “endogenous grid

¹³Subsidies to education are also guessed and updated to target public education spending when computing the baseline economies.

decision rules” are then interpolated back to the desired exogenous state space (b, i, θ) . As a preliminary to sketching the procedure it is convenient to rewrite the household decision problem as:

$$V(b, i, \theta) = \max_{c, s, h, i', x'} \{u(c) + \beta E_{z', \theta'} [V(b', i', \theta')]\}$$

where x' is defined as the non-stochastic portion of family wealth:

$$x' = Ra' + (1 + R^{-1}\kappa)(1 - \tau)wh.$$

The algorithm to compute decision rules is as follows:

1. Assume the borrowing constraint does not bind. Using first order conditions (FOCs), obtain $s(i, \theta)$ and $h(i, \theta)$ for each (i, θ) . These are always the unconstrained choices for s and h .
2. Guess initial derivatives of the value function $\left[\frac{\partial V}{\partial x'}\right]$ and $\left[\frac{\partial V}{\partial i'}\right]$ for state space (b, i, θ) .
3. Interpolate to compute $E\left[\frac{\partial V}{\partial x'}\right]$ and $E\left[\frac{\partial V}{\partial i'}\right]$ for exogenous grid of (x', i', θ) .
4. For each grid point in (i, θ, i') , compute the implied choice x' as follows:
 - (a) Combine the unconstrained FOCs for x' and i' and interpolate over the exogenous grid of x' to solve.
 - (b) Check if borrowing constraint binds. If so, combine the constrained FOCs for h and i' and interpolate to find constrained x' .
5. Use the human capital production function, FOC for i' , definition of a' , and budget constraint to compute (q, c, a', \hat{b}) for all points in (i, θ, i') . This produces decision rules for the state space (\hat{b}, i, θ) .
6. Interpolate the endogenous grid decision rules back to (b, i, θ) .

7. Compute decision rules where i' “binds” high.¹⁴
 - (a) Compute consumption for grid of x' , then interpolate to solve the FOC for x' .
 - (b) Check if borrowing constraint binds. If so, then interpolate to solve the constrained FOC for h to find constrained x' .
8. Update $\left[\frac{\partial V}{\partial x'}\right]$ and $\left[\frac{\partial V}{\partial i'}\right]$. Iterate from step 3 until convergence.

¹⁴Once state wealth b is sufficiently high, further increases will no longer result in increased early health spending (assets have a higher return).

APPENDIX C

PROOF OF PROPOSITION

Proof. For ease of exposition, I suppress controls (X, μ) and error terms (u) below. Due to collinearity, $Exp_{dc} + Exp_{dc}^* = K_c$, where Exp_{dc}^* denotes exposure during the three months after birth and K_c is some constant within each community. Denoting the total effect of prenatal exposure to food scarcity γ and the total effect of exposure during the three months after birth ϕ , the empirical specification can be derived:

$$\begin{aligned} Y_{idc} &= \alpha + \gamma Exp_{dc} + \phi Exp_{dc}^* \\ &= \alpha + \gamma Exp_{dc} + \phi (K_c - Exp_{dc}) \\ &= \alpha + (\gamma - \phi) Exp_{dc} + \phi K_c \\ &= \alpha + \delta Exp_{dc} + \theta_c \end{aligned}$$

where $\delta = (\gamma - \phi)$ and the constant K_c is absorbed by the empirical community dummies θ_c . The assumption that the effects of exposure to relative food scarcity are weakly negative during pregnancy and the three months after birth implies $\gamma, \phi \leq 0$. Together with the identity $\delta = (\gamma - \phi)$, this assumption implies that $\max \gamma = \delta$, or alternatively $\min |\gamma| = |\delta|$, where $|\cdot|$ denotes the absolute value. \square

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